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EYE LESIONS IN EXPERIMENTAL INFECTIONS SPECIAL REFERENCE TO ARTHRITIS*

CONRAD BERENS, M.D., D. MURRAY ANGEVINE, M.D., LOREN GUY, M.D., AND SIDNEY ROTHBARD, M.D.

Chronic uveitis and related inflammatory diseases of the eye, not caused by tuberculosis or syphilis, are a frequent cause of blindness, especially in adults (Cowan and Sinclair[1]), and present one of the most difficult problems in ophthalmology. Because the etiology of chronic inflammatory eye lesions is often in doubt, the experiments reported here were undertaken in the hope of elucidating some of these important problems.

The prevailing opinion is that many of these diseases are of infectious origin and are probably related in some way to a focus of infection that involves the eye either by metastatic invasion or by the elaboration of bacterial products. Such observations have been formulated mainly on clinical evidence, most of which has been hypothetical, and, if accepted as the final answer to the problem, will certainly retard rather than advance the search for the etiologic factors concerned in this important group of eye diseases. Great significance has been attached to the fact that a microörganism isolated from a sinus, tonsil, or tooth of a patient suffering with uveitis will react in a certain manner on laboratory media and will produce various lesions in animals. Most work of this nature has not been adequately controlled. Nevertheless, on incomplete and inconclusive clinical and experimental data, focal infection is considered by certain ophthalmologists as the chief etiologic factor in chronic uveitis.

It is also a general belief that iritis occurs as a complication in certain cases of rheumatoid arthritis and rheumatic fever. However, satisfactory statistics on the incidence of this type of iritis are not available. Dawson[2] reports that in his experience he has seen four cases of iritis in patients with rheumatoid arthritis, two of which resulted in loss of vision. Iritis was observed by Bauer[3] in 7 of 150 patients with rheumatoid arthritis. In a series of 200 cases of rheumatoid arthritis studied by Cecil and Angevine[4] there were 3 patients with iritis, an incidence of 2.5 percent. Both Swift[5] and Coburn[6] record iritis as occurring in rheumatic fever.

The etiology of rheumatoid arthritis and rheumatic fever is unknown although there is evidence that these diseases may bear a close relationship. This is based upon the studies of Swift,[5] Coburn and Pauli,[7] Nicholls and Stainsby,[8] Dawson, Olmstead, and Boots,[9] and others who have shown that the streptococcus, if not the etiologic agent, is possibly significant in both diseases.

Experiments were undertaken to obtain a clearer insight into the pathology

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of streptococcus arthritis in rabbits. Because the streptococcus has so frequently been considered as a cause of a considerable number of eye infections (Benedict, Von Lackum, and Nickel;[10] Brown and Dummer;[11] Rosenow and Nickel[12]) we believed that it would be of value to make a detailed and careful study of the eyes of these rabibts over a considerable period of time. Blood cultures were taken frequently during life and routinely at autopsy. The agglutinin and precipitin titers were determined at regular intervals. The sedimentation rate was determined repeatedly. Skin tests to determine sensitivity to streptococcus filtrate were performed in many instances.

To be certain that the eyes were normal, each animal was examined before injection. The eyes were observed almost daily for two weeks after the infecting dose was given and then at frequent intervals. Several detailed examinations of the fundi were made on each animal. When animals were killed, the eyes were sectioned. The paraffin method was used exclusively in order to obtain satisfactory cellular detail. If lesions were found on microscopic examination, additional sections were usually cut.

Several difficulties were encountered in these experiments. It is well known that the organs of many presumably normal rabbits, especially the liver, heart, and kidneys, contain accumulations of cells that may be wrongly interpreted as evidence of inflammation produced experimentally. The eye presented a similar problem. On microscopic examination an accumulation of cells, mostly lymphocytes and mononuclear leukocytes, was found at the corneoscleral junction of almost every eye. The extent of this accumulation of cells varied considerably in different animals. The possibility that an inflammatory process of similar nature may have occasionally extended to other structures must be considered.

The experiments have been divided into five groups:

Group I—Rabbits that received a single intravenous injection of one of several strains of streptococci.

Group II—Rabbits that received multiple intravenous injections of one of several strains of streptococci.

Group III—Rabbits that had been previously immunized either intradermally or intravenously with streptococcus vaccine and later injected intravenously with hemolytic streptococci.

Group IV—Rabbits in which we attempted to produce foci of infection at several sites.

Group V—Rabbits that received single intravenous injections of the following organisms: Staphylococcus aureus, Pneumococcus, B. typhosus, B. paratyphosus A, B. coli communior, Proteus vulgaris × 19, Brucella abortus, Brucella melitensis.

Pathology

Before entering into a detailed description of these experiments, the various types of pathologic lesions that have been produced will be briefly described.

1. An acute lesion that developed

Fig. 1 (Berens, Angevine, Guy, and Rothbard). The ciliary processes of a rabbit that had received 2.0 c.c. of a broth culture of B. abortus, 24 hours previously. The vessels are dilated, there is extensive edema, and the ciliary epithelium is destroyed. An occasional polymorphonuclear leukocyte and fine strands of fibrin can be seen. × 140.

Fig. 2 (Berens, Angevine, Guy, and Rothbard). The filtration angle in an eye of a rabbit that had received 1.0 c.c. of a broth culture of B. typhosus. There are considerable numbers of polymorphonuclear leukocytes in the anterior chamber, the canal of Schlemm, and the ciliary body. × 265.

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quickly and reached its height after about 5 hours and usually persisted for 24 or 48 hours was characterized by intense congestion of the vessels of the iris and ciliary body and occasionally by some clouding of the anterior-chamber fluid. Microscopically (fig. 1) there was edema of the ciliary processes and dilatation of the vessels associated in many instances with hemorrhage. The epithelial cells covering the ciliary body were often destroyed. In a few instances fine strands of fibrin were seen beneath the epithelium of the ciliary processes. There was also precipitated protein in the aqueous humor about the ciliary body and in the anterior chamber. In several instances (fig. 2) large numbers of polymorphonuclear leukocytes were seen in the posterior and anterior chambers, especially about the ciliary processes and in the region of the canal of Schlemm. This lesion was seen only in those animals that were killed within 48 hours or that died within a few days.

2. The lesion most frequently observed was an accumulation of lymphocytes, plasma cells, and mononucleur leukocytes in the ciliary body or processes (fig. 3) usually adjacent to the overlying epithelium.

3. In a few instances similar collections of cells were observed in the choroid (fig. 4) and less frequently in the iris (figs. 5 and 6).

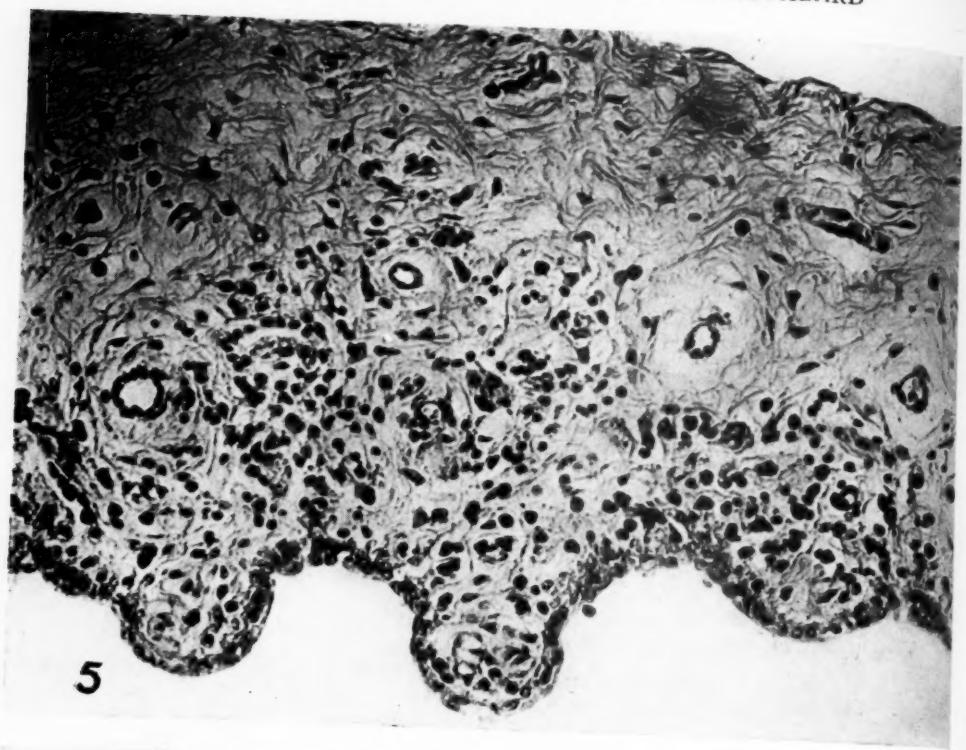
Group I

A group of 64 rabbits each received a single intravenous injection of one of several strains of streptococci. The dose of bacteria was usually 2 c.c. of an 18-hour broth culture, but in a few instances it varied slightly from this quantity. Thirty-five of these rabbits were injected with a strain of hemolytic streptococcus (Strain AB 13) obtained originally from the blood of a patient with rheumatoid arthritis. The eyes were examined frequently, and in two rabbits of this group there was a graying of the cornea. One rabbit developed this lesion six days after a single intravenous injection of 0.001 c.c. of hemolytic streptococci. The lesion was bilateral but most extensive in the right eye. The anterior-chamber fluid was aspirated. A few polymorphonuclear leukocytes and lymphocytes, but no bacteria, were seen in a smear of the fluid, and the culture was sterile. This lesion subsided after 13 days but became evident again after an intravenous injection of 1 c.c. of streptococcus filtrate. After 80 days the tension of the right eye was increased (31 mm. Hg with a Schiötz tonometer). There was also a slight dilatation of the vessels of the iris of one rabbit for a few hours after injection. There was no microscopic lesion of the eyes of this animal 60 weeks after injection. A few lymphocytes and plasma cells were seen in the region of the ciliary body in 5 of 35 rabbits.

Eleven rabbits received single injections of somewhat smaller doses of a hemolytic streptococcus (strain NY 5). There was congestion of the iris vessels in nine rabbits; in several instances it was intense. In the eyes of one animal, killed after four days, there was an infiltration of polymorphonuclear leukocytes in the

Fig. 3 (Berens, Angevine, Guy, and Rothbard). Lymphocytes and plasma cells in the ciliary body of the eye of a rabbit. The animal received 5.0 c.c. of the filtrate from a broth culture of a hemolytic streptococcus (strain AB 13). After 10 days, the animal received 2.0 c.c. of a broth culture of the same organism. The animal was killed 13 weeks after the last injection. × 260.

Fig. 4 (Berens, Angevine, Guy, and Rothbard). A collection of lymphocytes in the choroid of the eye of a rabbit that had received an injection (0.1 c.c.) of a broth culture of hemolytic streptococcus (strain AB 13). Another injection (1.0 c.c.) was given after 10 days. The animal died four days later. × 390.



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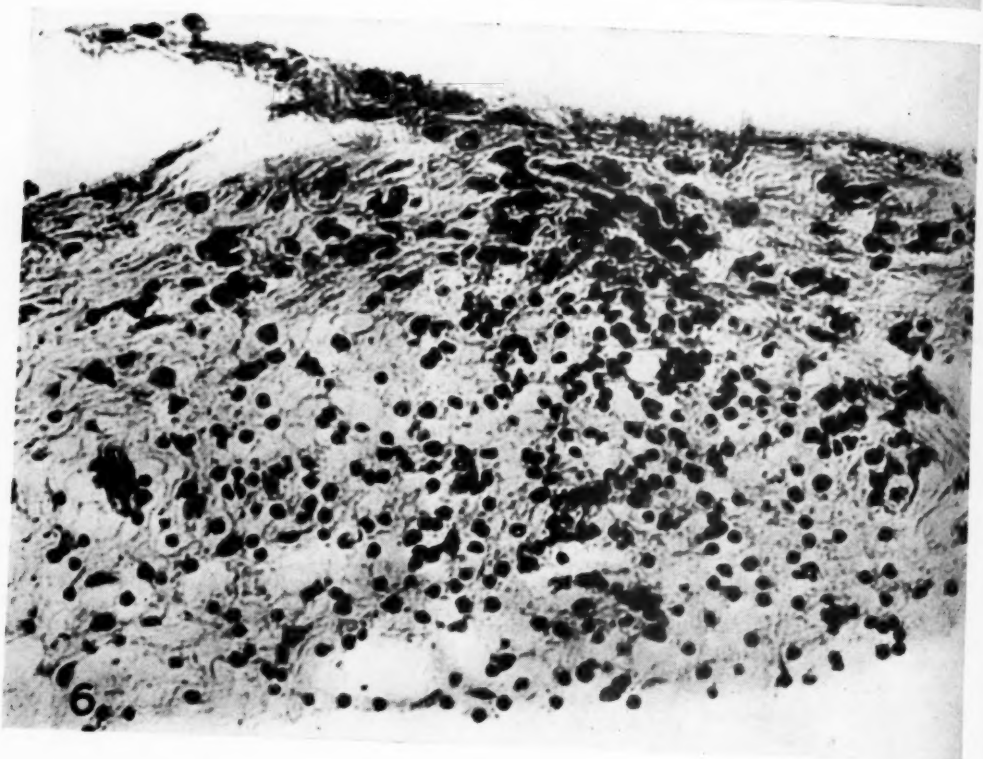

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angle of the anterior chamber in the region of the canal of Schlemm.

A strain of Streptococcus viridans freshly isolated from a case of subacute bacterial endocarditis produced no lesion in the eyes of two rabbits. Streptococci were obtained from the throat and teeth of several patients, most of whom were suffering with chronic uveitis, and were injected into 16 rabbits. In two instances the animals died within 24 hours, and there were polymorphonuclear leukocytes in the root of the iris of both eyes. There was also precipitated eosinophilic protein in the anterior chambers. Collections of lymphocytes and plasma cells were found in the ciliary body and choroid of three of the rabbits that lived for a longer period.

Of the entire group of 64 rabbits microscopic lesions were seen in the eyes of 13 (20 percent). In 3 they were acute, and in 10 they were of a more chronic nature.

Group II

Thirty-four rabbits received more than one injection of the organisms used in group I—namely, hemolytic streptococci (strains AB 13 and NY 5)—and other animals a Streptococcus viridans. Slight but definite congestion of the vessels of the iris appeared in only 3 of 26 rabbits that received strain AB 13 and microscopic lesions were found in the eyes of 2 of them. The vessels of the iris were congested in 3 of 4 rabbits injected with strain NY 5. No microscopic lesions weer found in the eyes of these rabbits. No visible changes were seen in the eyes of 4 rabbits that received injections of Streptococcus viridans. There was an infiltration of lymphocytes and plasma cells in the ciliary body, iris (fig. 5), or choroid (fig. 4) in 6 of 26 animals that had received injections of strain AB 13. Eight, or 24 percent of the entire group of 34 rabbits, showed this type of lesion.

Group III

Thirty-six rabbits were immunized by repeated injections of heat-killed streptococcus vaccine (strain AB 13) over a period of 50 days. One half of them were injected intradermally and developed a high degree of sensitivity to streptococcus filtrate. The others received the vaccine intravenously and acquired only slight sensitivity to the filtrate. Both groups of animals and a group of normal rabbits were injected intravenously with 2 c.c. of an 18-hour broth culture of hemolytic streptococci (strain AB 13). The rabbits were observed for a period of two months, and no gross eye lesions were found. The animals were killed, and the eyes studied histologically. A small number of lymphocytes and plasma cells were observed in the ciliary body in 2 of 7 intradermally immunized or hypersensitive rabbits (table 1). No lesions were seen in 9 intravenously immunized or in 9 control rabbits.

This experiment was repeated in essentially the same manner except that the animals received three intravenous injections of streptococci at intervals of 10 days. The first injection was 0.1 c.c., the second, 1.0 c.c., and the third, 2.0 c.c.

Fig. 5 (Berens, Angevine, Guy, and Rothbard). A diffuse infiltration of mononuclear cells throughout the posterior part of the iris. From the eye of a rabbit that had received at 10-day intervals injections of 0.1 c.c., 1.0 c.c., and 2.0 c.c., respectively, of a broth culture of hemolytic streptococci (strain AB 13). The animal was killed two weeks after the last injection. × 290.

Fig. 6 (Berens, Angevine, Guy, and Rothbard). A localized collection of lymphocytes and plasma cells in the iris of a rabbit that had received 0.2 c.c. of a broth culture of Staphylococcus aureus one week previously. × 400.

Hyperemia of the iris appeared in all of the intradermally injected rabbits within 5 hours after the second injection. It varied from a slight to an intense redness. No gross changes were observed in the intravenously immunized or in the normal rabbits. There were fairly extensive infiltrations of lymphocytes and plasma cells involving the iris, ciliary body, and choroid in the eyes of 9 of 12 intradermally immunized rabbits (table 1). Similar but less extensive lesions were seen in 3 of 8 intravenously immunized rabbits and in 5 of 12 normal rabbits.

TABLE 1

THE INCIDENCE OF EYE LESIONS PRODUCED IN NORMAL AND IN IMMUNIZED RABBITS BY MEANS OF SINGLE OR MULTIPLE INJECTIONS OF HEMOLYTIC STREPTOCOCCI (STRAIN AB 13)

Group	Rabbits	Eye lesions
Single Injections		
Intradermally immunized	7	2
Intravenously immunized	9	0
Controls	9	0
Multiple Injections		
Intradermally immunized	12	9
Intravenously immunized	8	3
Controls	12	5

A group of 8 rabbits received an intravenous injection of 5 c.c. of a streptococcus filtrate and 10 days later an intravenous injection of 2 c.c. of broth culture of hemolytic streptococci. There was congestion of the vessels of the iris in 4 rabbits. Collections of lymphocytes and plasma cells were seen in the choroid of one and in the ciliary body (fig. 3) of 2 rabbits, killed, respectively, 10, 25, and 89 days after injection.

Group IV

Using a hemolytic streptococcus (strain AB 13), we attempted to establish localized foci of infection at various selected sites in 74 rabbits. The dose was usually larger than that used for intravenous injection. In some animals single foci, and in others multiple foci were established. The following methods were used:

1. Suspensions of bacteria were injected into the following structures: the gum, accessory nasal sinus, prostate, testis, anterior chamber of the eye, pleural and peritoneal cavity, knee joint, and skin.

2. Segments of the uterus and Fallopian tube were isolated by ligature and injected with streptococci.

3. The renal pelvis was injected after ligation of the ureter.

Pledgets of cotton were soaked with streptococci and packed firmly into the nares, where they remained for several weeks.

The eyes were observed frequently and in only two of the entire group of animals was any dilatation of the vessels of the iris noted, this being slight. Organisms had been injected into the gums of both of these animals. No lesion was observed microscopically in sections of the eyes of these animals 60 days after injection. At the time of autopsy the focus was cultured to determine if the injected organisms were still present; in most instances it was sterile. We were successful in establishing a focus in the eye from which we were able to recover organisms for as long as 30 days after a single intraocular injection. There was infiltration of lymphocytes and plasma cells in the eyes of 9 of the 74 rabbits or in 12 percent (table 2). Only one of the animals with an eye lesion had arthritis.

Group V

It seemed desirable to observe the effect of various other strains of bacteria on the eyes and joints of rabbits. Small doses (0.1 to 2.0 c.c.) of 18-hour broth cultures of the following organisms were injected intravenously into 83 rabbits: Staphylococcus aureus, Pneumococcus (types I and XIV), B. typhosus, B. paratyphosus

A, B. coli communior, B. proteus, Brucella abortus, Brucella melitensis. Congestion of the vessels of the iris was observed within 2 to 5 hours after injectio in all the rabbits with the exception of 3 of 6 rabbits that received Brucella melitensis and 1 of 10 that received pneumococci. This reaction reached its height after 18 hours and had usually disappeared within 48 hours. The degree of redness varied from a slight erythema to an intense purple or maroon color. The

TABLE 2

THE INCIDENCE OF LESIONS PRODUCED IN THE JOINTS AND EYES OF RABBITS WITH INJECTIONS OF HEMOLYTIC STREPTOCOCCI INTO VARIOUS SITES

Site injected	Rabbits	Arthritis	Eye lesions
Gum	10	4	0
Accessory nasal sinus	11	3	3
Prostate and testis	11	2	2
Renal pelvis	3	0	2
Eye	6	1	1
Skin	6	0	1
Pleura	3	1	0
Joint	7	0	0
Uterus and tubes	6	0	0
Nose	4	0	0
Peritoneum	4	0	0
Gall bladder	3	0	0
Total	74	11–15%	9–12%

intensity of the reaction varied considerably with different organisms. B. proteus in doses of from 0.5 to 1.0 c.c. of a broth culture produced the most intense and constant changes. B. paratyphosus A, B. coli communior, and B. typhosus produced a similar reaction, but it occurred less regularly. Staphylococcus aureus, Brucella abortus, Brucella melitensis, and Pneumococcus caused only a moderate reaction.

A few animals died within 48 hours after injection and in these (fig. 1) edema of the ciliary processes associated with intense congestion of the vessels was usually present. In several instances there was associated hemorrhage into the surrounding tissue, and frequently destruction of the epithelium covering the ciliary processes occurred. The cytoplasm of many cells was either fragmented or absent, and the nuclei were usually pyknotic. Polymorphonuclear leukocytes, surrounded by fluid which stained pink with eosin (fig. 2), were often observed in the posterior chamber in the region of the ciliary body and in the anterior chamber. They were most numerous in the region of the canal of Schlemm. An acute inflammation of this nature was seen in rabbits that died shortly after injection, but most of the animals lived for 30 days and were killed. In only one of these (fig. 6) was a microscopic lesion seen in the eye. This was an accumulation of lymphocytes and plasma cells in the iris of an animal that had received a single injection of Staphylococcus aureus 30 days previously. The incidence of microscopic eye lesions in the entire group of 83 rabbits was only 6 percent.

The number of animals that developed eye lesions, which varied considerably in the different groups, is shown in chart 1. The following points are illustrated: (1) More lesions were produced in the first four groups by the strains of streptococci that were used than by other organisms. (2) The lesions were produced somewhat more frequently in immunized than in control animals.

Lesions in the eye were seen more frequently in the ciliary body or processes and less often in the iris and choroid (chart 2). In a few instances these lesions involved more than one structure. However, lesions of the uveal tract with the characteristics of chronic uveitis were observed. A large number of lesions were found six weeks after infection because the majority of the animals were killed after this interval. It is not possible to say what percentage of these animals would have had microscopically demonstrable inflammation in the eyes had they

lived for a longer period of time.

An attempt was made to correlate arthritis in the various groups with the eye lesions (chart 3). Animals with arthritis had no more eye lesions than animals with no arthritis; that is, we were unable

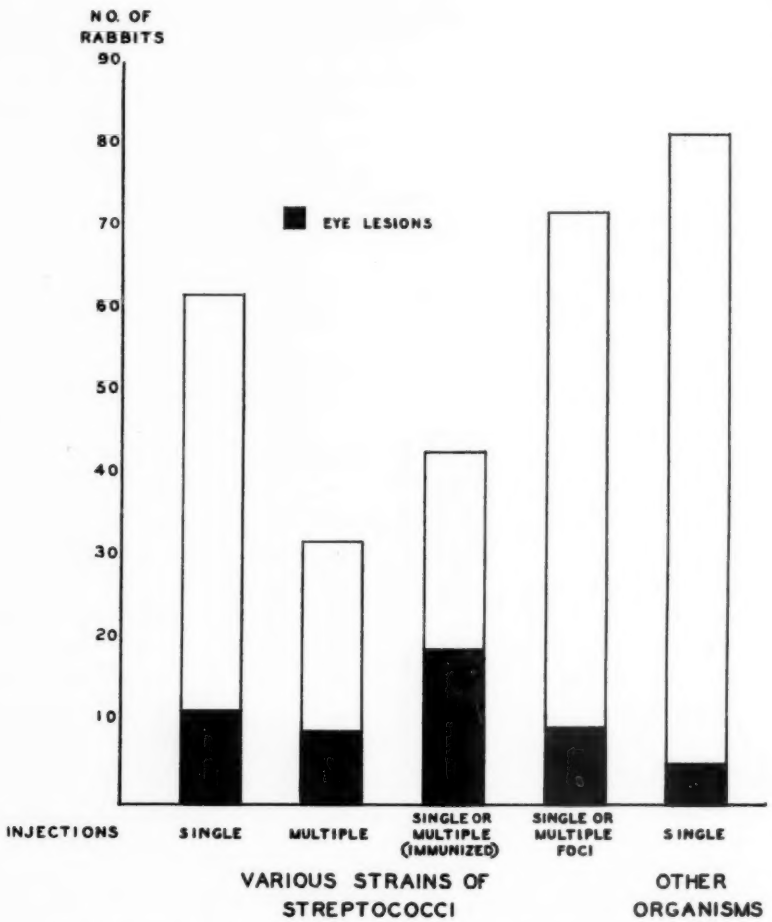

Chart 1 (Berens, Angevine, Guy, and Rothbard). The incidence of eye lesions in the various groups.

to demonstrate any relationship between the involvement of the eyes and joints in the experiments just described.

Discussion

Several strains of bacteria have been used to produce inflammatory lesions in the eyes of rabbits. These lesions are chiefly of two types: (1) an acute inflammatory process in the region of the canal of Schlemm with edema of the ciliary body and dilatation of the vessels of the iris, and (2) small accumulations of lymphocytes and plasma cells in either the ciliary body, choroid, or iris.

Rosenow and Nickel[12] and others have described lesions of the former type. Most of the experiments in which eye lesions have been produced by bacteria have been made with streptococci and staphylococci, and little work has been done to determine the effect of other organisms on

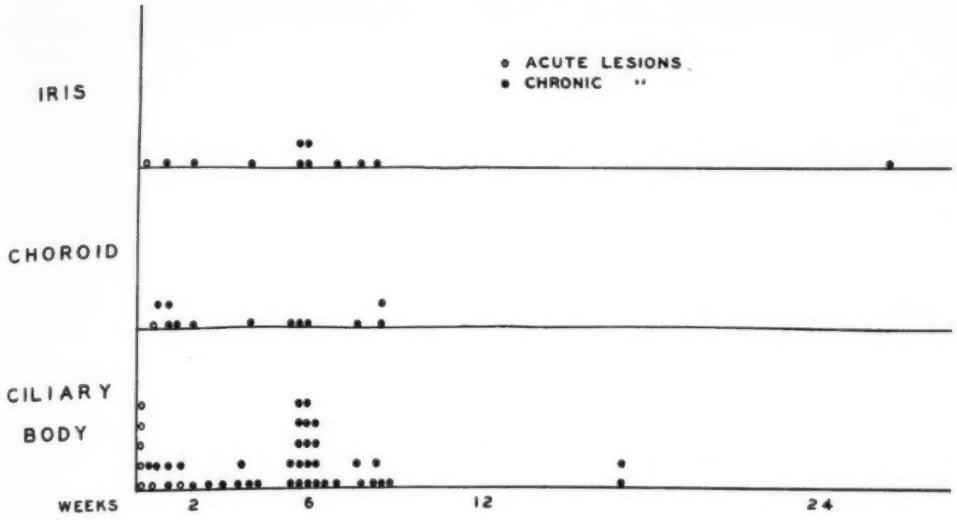

Chart 2 (Berens, Angevine, Guy, and Rothbard). Distribution of acute and chronic eye lesions.

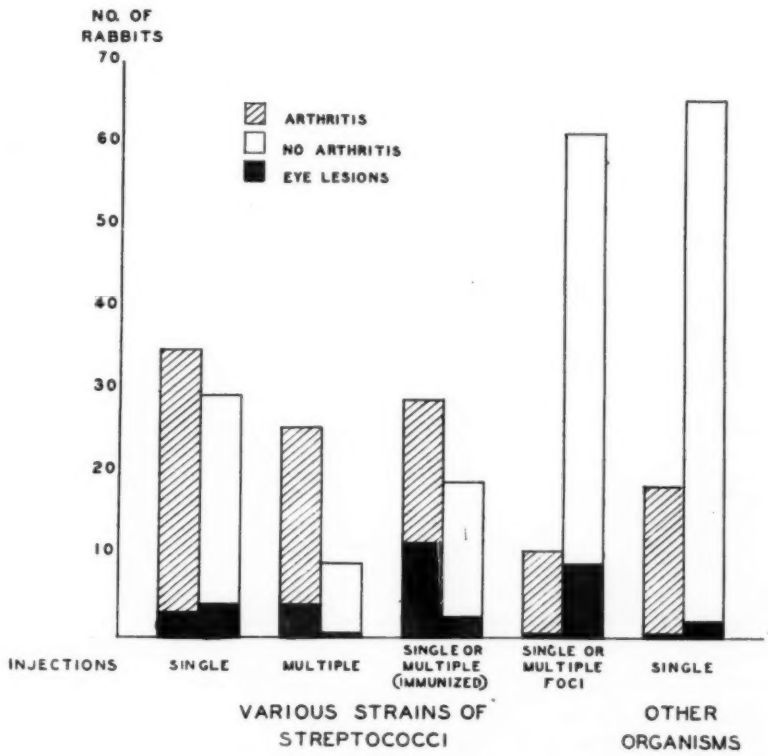

Chart 3 (Berens, Angevine, Guy, and Rothbard). Eye lesions in relation to arthritis.

the eye. In general, however, larger doses were used, and most of the animals were killed soon after injection so that no comparison of results is possible. The doses that we have used were smaller but sufficient to produce lesions in the joints and endocardium of a considerable number of animals. Several freshly isolated cultures from each of 9 patients with acute or chronic uveitis were injected intravenously in doses of from 2 to 5 c.c. into 19 rabbits. These organisms from patients with eye diseases produced no more eye lesions in rabbits than strains from other sources. These findings are in accord with those of Berens, Nilson, and Chapman,[13] who found that congestion of the vessels of the iris was produced as readily with organisms from patients who had no inflammatory eye disease as from those with acute or chronic iritis and uveitis.

From our studies it appears that certain strains of bacteria are capable of producing more extensive lesions in the eye than others. Of the hemolytic streptococci used, strain NY 5, a strong toxin producer, was more effective than AB 13, an organism that produces little or no erythrogenic toxin. Of the other organisms used, B. proteus, B. typhosus, and B. paratyphosus A produced the most extensive eye lesions. Additional studies are in progress to gain insight into the pathogenesis of this acute inflammatory process both from the standpoint of causative organism and host.

The incidence of microscopic lesions (chart 1) produced in the eye by multiple injections was 24 percent as contrasted with 20 percent in those receiving single injections. This difference is not significant because the number of animals infected with various organisms varied considerably in both groups. However, in a smaller group of animals in which one strain of streptococcus (strain AB 13) was used (table 1), the findings were significant. No lesions were found in the eyes of 9 rabbits that had received a single intravenous injection of organisms, but 5 of 12 rabbits (41 percent) developed eye lesions when multiple injections were given.

There were microscopic lesions in the eyes of 28 percent of the rabbits intradermally immunized with heat-killed streptococci that had received one infecting dose of organisms and in 75 percent of a similar group that had been infected with multiple (three) injections. The lesions in the sensitized group were more conspicuous than those seen in the control animals. There were fewer lesions in the eyes of the intravenously immunized than in the normal animals. The results of our experiments are in general agreement with those of MacLean,[14] who demonstrated a higher incidence of eye lesions in rabbits previously infected with Streptococcus viridans than in normal controls. In his experiments he scarified the cornea of certain animals and stated that trauma might have been an added factor in the production of the lesions. In our experiments none of the eyes were traumatized.

A group of 74 rabbits was infected with hemolytic streptococcus (strain AB 13) at various sites, and eye lesions were observed in only 12 percent of the animals. The percentage was considerably less than when the injections were given intravenously.

In the group that received single intravenous injections of other organisms, a microscopic lesion of a chronic type was observed in one of the 83 animals injected. The other lesions (chart 1) were acute and were present only in rabbits that died after one or two days. Because of the small number of lesions produced in these 83 rabbits, we believe that the

group may be considered as an index of the small amount of inflammation normally present in the uveal tract of rabbits.

Summary and conclusions

1. Acute or chronic inflammatory lesions have been produced in the eyes of rabbits by intravenous injections of relatively small doses of the following organisms: Streptococcus hemolyticus, Staphylococcus aureus, B. typhosus, B. coli, B. proteus, and B. abortus.

2. Various strains of streptococci, when injected intravenously, have produced more microscopic lesions of the uveal tract than any other organisms used.

3. Multiple injections of streptococci were considerably more effective in producing lesions in the eye than single injections.

4. The inflammatory lesions that were produced in a group of intradermally immunized (sensitized) rabbits were more frequent and extensive than in intravenously immunized or normal rabbits.

5. Few eye lesions were produced by means of foci of infection established in various sites.

6. The incidence of eye lesions was no higher in rabbits in which arthritis occurred than in those with no demonstrable involvement of the joints.

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Discussion

Dr. Frederick H. Verhoeff (Boston): I should like to ask Dr. Berens whether or not he obtained in any instance a condition similar to what we see in the clinical cases. I understand that all he obtained clinically was a little hyperemia of the iris which disappeared shortly, and microscopically the lesions he found were very insignificant. In the numerous injections did he never obtain

an instance in which the eye showed a condition similar to that of serious cases we see clinically with marked precipitates on the back of the cornea?

DR. BERENS: To my mind we have not produced experimentally the type of lesions that we see clinically. We do, however, have sections showing infiltrations with round cells which are somewhat similar to the type of lesion seen in patients with rheumatoid arthritis and rheumatic fever. We have not produced that chronic, recurring type of uveal inflammation which is observed clinically and which produces so much blindness.

DR. VERHOEFF: I should like to ask if he took an equal number of animals and made an equal number of microscopic examinations, whether he wouldn't find little lymphocytic nodules in the uveal tract.

DR. BERENS: I will ask Dr. Angevine if he will answer the question because he has done most of the experimental work.

DR. MURRAY ANGEVINE: In examining the eyes of these animals, we did find in practically every instance some infiltration of cells at the corneoscleral junction. This made the interpretation of some of the findings difficult; however, the cells were localized at the corneoscleral junction, and it did not seem feasible to examine the eyes of three hundred control rabbits because we did not feel that we would gain any information from such a study. However, in only one instance, in one group of animals that were not infected with streptococci, and not kept for a long period of time, did we find an accumulation of cells, other than those described. In that case there was an accumulation of cells in the iris. We feel that a study on any small group of animals would mean nothing, but on a comparative basis we feel that some of the figures presented are significant.

DR. WILLIAM L. BENEDICT (Rochester, Minn.): I should like the authors to elucidate their meaning of "sensitization" and "immunization" in these experiments.

DR. ANGEVINE: These two groups of animals probably should be considered as essentially the same; in other words, they could both be called, immunized; however, because of the work of Dr. Swift, especially on sensitization with streptococci, who showed that animals injected intradermally, developed a high degree of skin sensitivity to the streptococcal protein and were more sensitive than animals injected intravenously with a similar protein, we felt from the standpoint of classification that it was more suitable to separate our groups of animals in this manner. The intradermally injected animals, however, were much more sensitive at the time of infection than were those that had been injected intravenously.

When bacterial protein is injected intravenously over a long period of time, the animal is probably being continually desensitized, but if no injections are given for some time, the animal may become highly sensitive.

CHAIRMAN JACKSON: In presenting this subject to us, Dr. Berens referred to Dr. Dawson. He is present and we would be very glad to have his comments or questions that he may raise. (Applause.)

DR. M. H. DAWSON (New York): The essentially "rheumatic" nature of certain forms of iritis has of course long been recognized. The association of acute iritis and rheumatic fever is particularly well known. The iritis which occurs with rheumatoid arthritis is of a more stubborn nature and not infrequently leads to serious complications. For some curious reason iritis is perhaps more commonly seen in that type of rheumatoid arthritis which affects the spine; namely, Strümpell-Marie spondylitis. It is therefore of particular interest to those of us who are attempting to elucidate something of the

nature of rheumatic diseases to hear this discussion from those who are primarily interested in diseases of the eye.

The association between diseases of the eye and diseases of the joints has led certain anatomists and embryologists to speculate on the relationship between synovial tissue and the tissues of the ciliary body. This relationship is a very mysterious one, but I believe that we now have, for the first time, a clue as to its nature. In a paper before this Association Dr. Meyer will report that he has been able to isolate the same acid polysaccharide from both synovial fluid and vitreous humor. It is too early to know the significance of this observation, but we are hopeful that it may lead to further interesting discoveries.

I should like to thank you very much for the privilege of commenting on this most interesting communication.

LECTURES ON MOTOR ANOMALIES

IV. The etiology of strabismus*

A. Bielschowsky, M.D.
Hanover, New Hampshire

The question as to the origin of squint has been discussed for more than two centuries. Innumerable theories have been published, not one of them applicable to all the different kinds of strabismus. The oldest theories were based on the assumption of a displacement of one of the maculae, due to which the patients were supposed to choose a squinting position in order to get the images of the fixated object on both maculae and to see it single (De la Hire, 1743). That theory seemed to be substantiated by the so-called paradoxical diplopia arising after an operative correction of the deviation. We speak of paradoxical diplopia, for instance, in patients who did not notice diplopia as long as they had their convergent squint, but had crossed diplopia after a convergent squint of a rather high degree had been reduced to a small fraction of the former amount. We know today that this kind of diplopia has nothing to do with a congenital displacement of one macula, but originates from an anomalous sensorial relationship between the two eyes. Another old theory considered the inequality of the two eyes to be the cause of strabismus. According to Buffon (1743) the patient resorts to squint in order to avoid the disturbance arising from the blurred and distorted retinal images of the weaker eye. One remembers this old theory of Buffon's in view of van der Hoeve's theory of diplopia-phobia as one of the causes of strabismus. When Dieffenbach (1840) had, for the first time, straightened the eyes of strabismic patients by myotomy, a disproportion between the muscular functions due either to abnormal insertion or abnormal structure was believed to be the true or main cause of strabismus. The investigation of the problem was considerably advanced by Donders's discovery (1863) of the intimate relationship between accommodation and convergence, and of the role played by

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*From The Dartmouth Eye Institute, Dartmouth Medical School. Read before the Seventh Annual Mid-Winter Clinical Course of the Research Study Club, Los Angeles, California, January, 1938.

hyperopia in the etiology of convergent strabismus, owing to the excessive accommodation required in hyperopia to obtain sharp retinal images. To be sure, Donders realized that convergent strabismus is to be met with in only a comparatively small number of hyperopic eyes, and that the same amount of hyperopia may be found in people with and without squint. He ascertained that the chance of acquiring convergent squint increases in proportion to the inferiority of the vision of one eye. As long as both eyes have equal vision the tendency to see single is strong enough to loosen, within certain limits, the functional connection between accommodation and convergence, so that the visual lines may be kept parallel, in spite of an accommodative effort to overcome hyperopia up to 3 or 4 diopters. Likewise, an absence or reduction of accommodation due to myopic refraction is, according to Donders, the starting point in the etiology of divergent squint. Donders's doctrine has been substantiated by those cases in which convex glasses correcting an existing hyperopia cause the convergent strabismus to disappear. On the other hand, grave objections have been made to the overestimation of the part played by refractive errors in the etiology of squint. The main objections are as follows: 1. The great majority of people with refractive errors do not squint. 2. Among the patients with convergent squint there are numerous cases of very slight hyperopia as well as of emmetropia, and even myopia; on the other hand, there are a great many cases of divergent strabismus with emmetropic or hyperopic refraction. 3. As a rule, the angle of squint does not correspond to the excess or lack of accommodation required because of hyperopia or myopia. Usually, at least in convergent strabismus, the deviation is much greater than it should be if the excess of convergence were just proportionate to that of the accommodation compensating the hyperopia. As to divergent squint, although about 50 percent or more of such patients are, indeed, myopic, nevertheless a complete lack of accommodation will cause only a *relative* divergence; that is, an insufficiency of convergence in near vision, but not necessarily an absolute divergence for distance. The partisans of Donders's doctrine have not been able to refute the above objections satisfactorily. Taking into consideration the fact that the relation between accommodation and convergence can be adapted to a great extent to the requirements of binocular single vision in spite of refractive errors, the question arises: Why, in a small percentage of hyperopic eyes (8 to 9 percent), notwithstanding fairly good and equal vision of both eyes, does that so-called relative range of accommodation fail to effect sharp as well as binocular single vision?

Hyperopia does, indeed, play an important part in the etiology of convergent strabismus, but does not cause a *permanent* squint even in persons in whom the fusion tendency is weakened or suspended by unilateral amblyopia. Only a *periodic* squint will arise in hyperopic persons who have equal vision in both eyes, if the amount of hyperopia exceeds the amplitude of the relative accommodation, and the excessive accommodative effort necessary to overcome the refractive error cannot be reconciled with a convergence innervation required by the distance of the fixated object. So long as the hyperopic person does not mind indistinct vision the eyes will be parallel in spite of the hyperopia, provided there is no anatomical factor that produces the cross-eyedness. Uncorrected myopia may be responsible for an insufficient convergence innervation but

will cause a divergent squint for *distance* no more than would a complete loss of the convergence movement in cases of convergence paralysis, if the ocular position of rest is orthophoria. Thus we may take it for granted that ametropia cannot cause a permanent squint unless there is at least one assisting etiologic factor.

According to Schweigger and Alfred Graefe, either the mechanical elastic predominance of one group of muscles or an insufficiency of the antagonistic group must be considered as the main cause of strabismus. Stilling and Landolt claimed that squint will develop only in patients having either a convergent or divergent position of the eyes due to topographic anatomic—that is, mechanical—factors when binocular fixation is abolished. French and English authors, such as Panas, Parinaud, and Priestley Smith, in discussing the origin of squint, lay the main stress on a disturbance of the cortical nervous centers governing binocular single vision. Panas, for instance, considered convergent strabismus to be a purely functional anomaly, in which neither the peripheral nerves nor the ocular muscles were affected. He referred to the disappearance of convergent squint during sleep and narcosis, moreover, to the role played by heredity, and particularly the neuropathic constitution, in the etiology of squint. C. Worth and many of the modern authors are inclined to minimize the importance of refractive errors, muscular imbalance, and other factors. In the opinion of C. Worth, the decisive factor is a congenital deficiency of the fusion apparatus, or one acquired in earliest childhood.

Thus it must be confessed that up to this day the etiology of squint has not been cleared up satisfactorily. However, the further investigation of our problem may be started with some unequivocal facts. The factors which play a part in the etiology of strabismus may be divided into three groups, according to their different nature. The faulty position of the eyes relative to each other may depend on mechanical conditions, such as the anatomic relations between the eyeballs and their adnexa, on the physical qualities of the latter, particularly of the fascia and the muscles, on their elasticity, length, insertion and so forth, on the volume and the quality of the tissues behind the eyeballs, and finally on the size and form of the orbits and the bulbi. In a second group the mechanical conditions may be perfectly normal but an excessive or spastic motor innervation may produce squint of one kind or another. In a third group both mechanical and nervous factors may be combined in causing a squint. But, as a rule, all these factors will produce merely a latent squint so long as the fusion apparatus is strong enough to overcome the tendency to squint. The latent squint may become temporarily manifest if the efficiency of the fusion apparatus is reduced by fatigue, exhaustion, or narcotics. If the fusion faculty is lost, a *periodic* will be transformed into a *permanent* squint. Let us consider the cases belonging to the first group. Only a small fraction of them shows a *permanent* squint due solely to mechanical factors, such as adhesions between the eyeballs and the orbital bones caused by injuries, or by an anomalous muscular structure, either of congenital origin or acquired during life as a result of lesions, inflammations, or tumors. In most cases in the first group the tendency to squint cannot be derived from a specific mechanical trouble, since there can be found neither a marked restriction nor an excess of any ocular movement. The only proof that the anomalous position of rest in those cases is based on mechanical conditions may be obtained by exclud-

ing, as far as possible, those innervations which may affect the position of the eyes relative to each other; for instance, by correcting any existing hyperopia and suspending the influence of the fusion apparatus. We know that in more than 80 percent of normal persons of different ages and sex, the position of rest is not orthophoria but some kind of heterophoria that occasionally manifests itself for a shorter or longer period if the fusion faculty is weakened. In all such cases the squint will become permanent when the fusion faculty is definitely lost. I* have made an extensive study of 289 patients whom I had known when their eyes were normal and coöperated perfectly in binocular vision. After the latter was lost because one eye had become highly amblyopic or blind as a result of an injury or an illness, or after a cataract had been removed, strabismus developed; in 70 percent of all the patients it was a divergent squint, in 50 percent amounting to more than 6 degrees; in 10 percent divergence was combined with a vertical deviation. Convergent strabismus developed in only 10 percent of my patients after the loss of binocular vision. But by comparing the frequency of the different kinds of squint in people who had lost fusion in an earlier stage of life with that in persons who had lost it later on, I could ascertain that in children under 15 years convergent squint developed four times as frequently as in people who lost binocular vision after they were 30 years of age, a proof of the gradual change of the position of rest and of the fact that during adolescence convergent squint will, not infrequently, disappear spontaneously. From these statements we must infer that an anomalous position of rest, that is to say, a heterophoria or latent squint based on anatomic-mechanical conditions, may temporarily become a *manifest* strabismus but never a *permanent* one, so long as the functioning of the fusion faculty is intact. The same is true in cases of the second group, in which strabismus is caused either by abnormal innervations, for instance, by an excessive convergence innervation arising when an excessive accommodation in hyperopia is required to get clear retinal images, or in cases of double hyperphoria, which is produced by involuntary spasms of the vertical divergence innervations. In all these cases the temporary or periodic squint will not become permanent so long as the fusion apparatus is strong enough to overcome the tendency to squint. As to the cases of typical relative hyperopia, as it was called by Donders, a permanent squint will not arise even if the fusion tendency is lost because of monocular amblyopia or blindness. Those patients show convergent squint only temporarily; namely, when they try to get clear retinal images by an excessive accommodative effort necessary to overcome their hyperopia. The presence of a permanent convergent squint in such cases would always suggest that there is an anomalous position of rest as a third etiologic factor in addition to both hyperopia and the loss of fusion. Even in the majority of cases belonging to the third group, in which both mechanical and nervous factors produce the tendency to squint, there will be only a periodic squint unless a third factor—namely, the loss of fusion—came to be associated with the two other factors. The above rules are not valid unconditionally. First, it is obvious that it depends on both the kind and the degree of the anomalous position of rest whether or not it can be overcome by an adequate fusion innervation, the range

* Bielschowsky, A. Ueber die relative Ruhelage der Augen. Ber. ü d. Versamml, d. ophth. Gesellsch., Heidelberg, 1913, v. 12, p. 67.

of which, although varying considerably in different individuals, is limited. Second, it may depend on the kind of work the patients are doing and on the time they must give to it whether or not a periodic squint due to both an anomalous position of rest and excessive motor innervations may be transformed into a permanent strabismus. That may occur in such a way that a contracture of a muscle or of a group of muscles which, in the beginning, is functional—that is, contraction and relaxation, respectively taking place according to circumstances —may become fixed by a gradually developing change in the muscular structure. This would occur the more easily the longer the functional contracture were maintained by an excessive innervation and the resultant squinting position of the eyes were assisted by mechanical factors which favor the same kind of deviation.

The momentous role played by deficiencies of the fusion apparatus in the etiology of squint must, indeed, be acknowledged unhesitatingly. On the other hand, there is no doubt that even a complete lack of the fusion mechanism cannot cause permanent strabismus in cases in which there is a normal position of rest; that is, orthophoria. They will present an insufficiency of convergence in near vision if the refraction is emmetropic or myopic. Or, individuals with uncorrected hyperopia may have a *periodic* concomitant squint for distance, as well as for near, because of the absence of the relative range of convergence. But there is no squint in spite of the lack of fusion so long as they do not mind indistinctness of the retinal images while their hyperopia is not overcome by accommodation.

In most cases of strabismus it is not very difficult to find out which and how many of the main factors first discussed are responsible for the anomaly. If there is any doubt as to whether abnormal innervations take part in the development of a squint, the angle of deviation must be measured several times under equal conditions: the more constant the angle of squint the more likely it is that the deviation originates from an anatomic-mechanical basis. Unsteadiness of the angle of squint points to abnormal, especially excessive or spastic, innervations as an essential etiological factor. Such innervations may be intermittent, since spasms come and go at irregular intervals, with more or less complete relaxation. However, even if the spasms are relatively constant, there will never be a constancy comparable to the steadiness of the deviation found in cases in which the deviation is based solely on anatomic-mechanical or on paralytic conditions. This will be substantiated, of course, if the deviation is measured repeatedly, but always for one and the same—usually the primary—direction of gaze, because there is no case of concomitant squint in which the angle of the deviation does not show slight variations in different directions of gaze; for instance, a divergent strabismus, will, as a rule, increase in the upper part of the field of fixation while it decreases in the lower part, due to physiologic-mechanical conditions favoring divergence in elevation and convergence in depression of the gaze. Furthermore, in most cases there is a slight decrease in the angle of both convergent and divergent deviations in extreme dextro- as well as levoversion, because the associated external and internal recti even under normal conditions do not respond equally to the impulses.

The unsteadiness of strabismic deviations just discussed must not be misconstrued as proof of a paralytic origin, for the latter causes the angle of squint to differ according as the paralyzed

muscle must participate, more or less, or not at all, in a voluntary movement. In a recent case of paralysis the angle of squint will be found to be fairly constant if measured at different times for one and the same direction of gaze, and its increase, respectively decrease in other directions will likewise be found to be constant in repeated tests, in contrast to the unsteadiness of the deviation due to spastic innervations. On the other hand, one must keep in mind that a paralytic deviation does not always retain its typical features indefinitely. There are many cases in which the paretic muscle regains its function partially or completely without a corresponding decrease of the deviation, because, while the muscle is recovering, a contracture of the antagonist of the paretic muscle takes place. If that contracture is due solely to an increased tonus, it will gradually decrease and finally disappear, after the pathologic origin causing both the paresis and the functional contracture has been removed. Otherwise, the functional contracture may be transformed into an organic one, due to a change in the muscular structure which can no longer be restored spontaneously. In either case, the deviation maintained by a temporary functional or a permanent organic contracture will not show the original paretic characteristics but those of a concomitant squint, the paretic origin of which can only be surmised, but is no longer evident, from the history, particularly from the sudden and simultaneous appearance of diplopia. Such cases are very instructive in the consideration of the problem in which we are interested because they demonstrate that a deviation of exclusively nervous origin may, in the course of time, either disappear or become fixed, displaying the behavior of a squint based primarily on mechanical conditions.

In only a small minority of cases may the paretic origin of a concomitant squint be inferred from the history, particularly from the statement of a sudden onset of the squint combined with diplopia. There are, likewise, only very few cases of true concomitant squint in which the deviation can be shown to be a congenital anomaly. Congenital deviations, almost without exception, show either deficiencies or irregularities of certain ocular movements such as never occur in concomitant squint. On the other hand, there is no doubt that in numerous cases the development of squint is favored, or even facilitated, by a congenital disposition. The great part played by heredity in the etiology of strabismus is substantiated beyond doubt by many statistics. I, personally, know some families in which both parents and all the children were cross-eyed. If only one of the parents squints, at least one of the children, as a rule, will be found to have the same or some other type of strabismus. Very often, indeed, the parents do not squint, but it will be found that one of their brothers, sisters, nephews, or nieces may squint. Moreover, heredity cannot be excluded if nobody in the family is cross-eyed. Carefully compiled statistics have shown that among the nonsquinting relatives of a squinting child, high ametropia, anisometropia, monocular amblyopia, heterophoria, or weak fusion faculty is to be met with much more frequently than in families which have no squinting member; that is to say, the offspring of people affected even with one or two of the anomalies that play a part in the etiology of strabismus have a much greater chance of acquiring the latter than do other children. A general weakness of the central nervous system, a so-called neuropathic condition, manifesting itself in an abnormal excitability, combined with an abnormal exhaustibility, is found

rather frequently as an inherited anomaly in children with convergent strabismus. I am not inclined to consider a neuropathic constitution as an important factor in the etiology of ordinary concomitant strabismus, because there are so many squinting children who not only show no signs and symptoms of neuropathic origin but, in addition to excellent physical conditions, a normal, or even an unusually high degree of intelligence. However, I do not hesitate to admit that the main characteristics of neurasthenia, particularly the combination of abnormal excitability and exhaustibility, are frequently at the bottom of either spasms or insufficiencies of certain innervations, particularly of the convergence, causing *temporary* deviations which are frequently, but erroneously, included in heterophorias. Furthermore, there is no doubt that neuropathic individuals with a slight heterophoria suffer from various subjective troubles, such as headaches, dizziness, diplopia, and so forth, whereas people with a normal nervous system have no symptoms whatever as a result of the same or even a higher degree of heterophoria.

Some authors consider strabismus a stigma of degeneration relating to an allegedly frequent combination of strabismus and stuttering, left-handedness, and other signs of abnormal development. According to my experience the above signs of degeneration or of faulty development are no more frequent in squinting individuals than they are in nonsquinting individuals.

From the history of many cases we learn that convergent squint was noticed after the occurrence in early childhood of one of the general infectious diseases, such as measles, scarlatina, whooping-cough, or other disorders that cause temporary affections of the brain. Considering that the center of the fusion apparatus is one of the most sensitive and vulnerable parts of the cortex, the function of which may be inhibited by mere fatigue or by a psychic shock, it seems very likely that a slight meningitis may cause lasting insufficiency or a total loss of the fusion faculty, depriving patients with a latent squint of the most effective defense against the transformation of a heterophoria into a manifest and permanent strabismus.

Aniseikonia, that is, the difference in the size and shape of the ocular images, seems to play an important part in the etiology of strabismus. This is suggested by the fact that a difference of five or more percent cannot, as a rule, be reconciled with binocular single vision, people with periodic or permanent squint showing, very frequently, an unusually high amount of aniseikonia.

In the discussion of the treatment of squint I shall revert to these interesting findings of Ames and his collaborators. Careful attention must be paid to all the details in the case history of any squint, since it might give valuable clues to the possible origin of the anomaly in the individual case, and therefore also to a rational therapy.

The examination of strabismus

As to the examination of squint, I refer, first, to what has been said in the discussion on heterophoria, particularly about the necessity to start, in every case, with objective tests for the motility of the eyes and for the kind and amount of the deviation. Most important, though sometimes putting the patience, perseverance, and skill of the doctor to a hard test, is the examination of very young children. The kind and the approximate amount of the deviation can be easily ascertained. Somewhat harder is the determination of the possible ametropia, which must be examined by reti-

noscopy after atropine has been given for at least three successive days. It can be very difficult and requires a good deal of patience to find out whether the squint is of paralytic or of nonparalytic origin. The last, but very important task to be accomplished, will be to determine approximately the visual acuity of each eye. It must be ascertained whether either eye has foveal vision. Children, even during the first year of life, very quickly become accustomed to the glasses which, in some cases, cause the squint to disappear and prevent the development of an amblyopia ex anopsia. Credit must be given to Claude Worth for having ascertained that the faculty of foveal fixation in the permanently squinting eye of a little child can be lost very quickly but can, just as quickly, be restored within a few weeks or even days, by occluding the fixating eye for a couple of weeks, thus forcing the continual use of the squinting eye. The more advanced the patient is in age the more time it will take to improve the function of the squinting eye. Because of this fact one must control the vision of the squinting eye in the earliest possible stage of the squint, the more so as a prolonged occlusion is much easier to accomplish before the child must go to school. But Claude Worth's statement that after the sixth year of life an amblyopia due to disuse can no longer be improved, is erroneous. I have seen quite a few adults with a squint that has existed since earliest childhood and a high amblyopia which was improved to nearly normal vision after the vision of the normal eye had been lost or reduced to a minimum by injury or disease. Of course, in a certain number of cases the amblyopia of the squinting eye may be due solely or partially to an organic and irreparable disorder; as, for example, a high ametropia or anomaly of the refracting media or of the eyeground. But there are many cases in which even the most careful examination will not display any anomaly that would account for the amblyopia. From the fact that the latter can be improved frequently by exercise, one has to infer that the disuse of the squinting eye is the main or only cause of the amblyopia. We do not yet know why an eye which may have had normal vision originally has become amblyopic or has even lost central fixation, while in other cases the vision of the squinting eye remains unimpaired; furthermore, why a squinting eye which has only a fraction of normal vision from the first, keeps that fraction unchanged during a period of many years. Neurologists know of an analogous disorder: in paralysis existing since birth or earliest childhood, true paralysis of the tactile sense has frequently been observed. For, in spite of the full integrity of the recipient organs as well as of the afferent pathways and the appertaining cortical area, no engrams are produced due to the disuse of the arm. If the patient is forced to use the hand for touching, the tactile paralysis disappears within a few days or weeks, but the new tactile engrams are unstable for a considerable time; they can be lost again unless the hand is used continually for touching. The tactile engrams are, as a rule, acquired during the first three years of life. No hemiplegia occurring after the third year will produce any astereognosis. In contradistinction to this functional astereognosis due to disuse, the tactile paralysis of organic origin—destruction of the postcentral gyrus or of the afferent pathways—can be improved very little, or not at all, by exercises.

The examination of squint in adults must be initiated by the determination of the refraction and visual acuity. If the movements of both eyes are equal

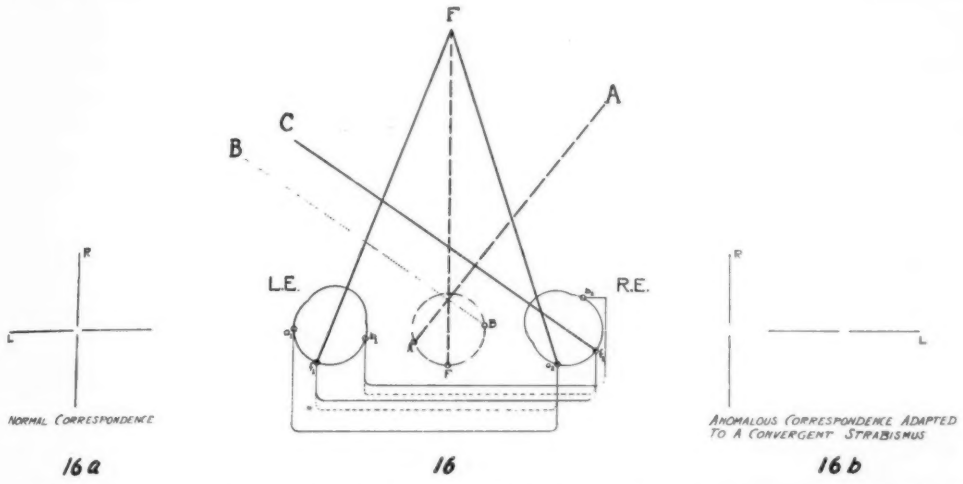
and not markedly restricted, the kind and the angle of deviation must be tested objectively, as exactly as possible, the more so since the diplopia test is not applicable in many cases. If the squinting eye lacks foveal fixation, the angle of squint may be measured on the perimeter, or opposite the little lamp forming the center of the tangent scale. While the lamp is fixated its reflection is seen lying in the periphery of the cornea of the squinting eye; then the patient is asked to look along the numbers of the scale until the reflection of the lamp has arrived at the center of the pupil or of the cornea of the squinting eye. The number fixated at this moment by the normal eye indicates the approximate amount of the deviation, not taking the so-called angle γ into account. This inaccuracy does not matter if the angle of squint is considerable because, due to the amblyopia, only a cosmetic result can be obtained by the treatment. If both eyes have foveal fixation an exact objective measurement can be accomplished in different ways: Either by means of prisms, transferring the eccentric image of the fixated light in the retina of the squinting eye from the periphery to the macula, so that after covering the fixating eye the other eye no longer moves when the patient is asked to look at the light, or by ascertaining that number on the tangent scale to which the squinting eye is directed while the other eye fixates the light in the center of the scale. If that number has been ascertained there is no movement of the squinting eye when the patient is ordered to fixate alternately that number and the light with the squinting, respectively, the other eye. In order to get the most precise measurement possible in patients with foveal fixation of both eyes, one may ask them to fixate for ten seconds with one eye the center of a glowing filament, which is either in a vertical or horizontal position, according to circumstances, while the other eye is covered. Then, with both eyes open the patient will be asked which number coincides with the negative afterimage of the glowing filament while the dominant eye fixates the light. The image of that number must be formed on the macula of the squinting eye, which is bearing the center of the afterimage. In most cases of concomitant squint the diplopia test is of no avail if it is a question of measuring the angle of squint either because of monocular amblyopia or because the innate retinal correspondence is not functioning. Spontaneous diplopia occurs only exceptionally, if the squint has arisen in early childhood, but is absent also in the great majority of cases where squint develops in adults. The absence of diplopia is due partly to a monocular amblyopia, partly to the so-called suppression of the retinal images of the squinting eye, partly to the development of an anomalous sensorial relationship between the two eyes.

It is obvious that the greater the amblyopia of the squinting eye the more readily will suppression set in. But even if both eyes have equally good vision, a monocular suppression of the eye that is squinting at a given moment is to be found in the great majority of cases. To understand the development of suppression one has to remember the physiologic rivalry between the two eyes. If different images are presented to the eyes in the stereoscope, the eye, the visual field of which is darker or contains less contours will be defeated in rivalry; that is, it will be suppressed for a longer or shorter period. This temporary monocular suppression makes it possible to use the ophthalmoscope or microscope without closing the second eye. The suppression ceases as soon as a stronger stimulation, for instance, a flickering or movement occurring in the visual field

of this eye, attracts attention. If different images are presented to the eyes—for instance, in the stereoscope—the eye the visual field of which is more monotonous than that of the other eye will be defeated in rivalry. Another factor that facilitates suppression in strabismus is, of course, the inadequacy of the image lying on a peripheral retinal area of the squinting eye, as compared with that of the same image situated on the macula of the fixating eye. The development of suppression is more difficult or even impossible if the angle of squint changes with every change of the direction of gaze. That accounts for the fact that there is no suppression, as a rule, in paralytic squint so long as it is nonconcomitant. But also in concomitant squint the suppression of the retinal images of the squinting eye is not equally strong in the different parts of the retina. The maximum of suppression is to be found in the area bearing the images of objects fixated with the other eye. In most cases, even in those with equal vision in both eyes, diplopia cannot be elicited merely by weakening the retinal images of the fixating eye. But there is no patient with strabismus who cannot be made conscious of double images if, in addition to the darkening glass put before the fixating eye, the retinal images of the squinting eye are displaced by a strong prism in one direction or another. It is significant that diplopia is noticed not only if the image of the fixated light is shifted toward the fovea but even if toward the periphery; that is, a place with a poorer function than that on which it was situated originally. Thus, although the retinal images of the squinting eye are suppressed under the ordinary conditions of seeing, the intensity of suppression varies within wide limits, and that not only in different individuals with the same kind and degree of squint, but also in one and the same patient according to the part of the retina of the squinting eye which is stimulated by the object fixated with the other eye. Sometimes the borders of regions with different degrees of suppression can be determined rather accurately, whereas in other cases they show a considerable variability. Suppression plays an important part in the investigation of the visual act as well as in the treatment of strabismus. Not until the suppression has been overcome is one able to investigate the sensorial relationship between the two retinas by means of the diplopia test.

When diplopia has been elicited, the position of the double images relative to each other will correspond only in a minority of the cases to the objective angle of squint plus the deviating effect of the prism. A patient with a convergent squint of 25 arc degrees, with a prism of 20 prism diopters, base out, in front of the squinting eye would, for instance, see homonymous double images with a separation of 15 degrees. But in the majority, the double images that arise after the prism has been put before one eye will not correspond to the objective angle of squint, but their position relative to each other would be approximately the same as in a nonsquinting person with binocular single vision if that prism were applied in the same position. In a case of convergent squint of 25 degrees with a prism of 20 prism diopters, base out, before one eye the double images would be crossed, the distance between them being approximately 10 (arc) degrees; that is, just as in a person with orthophoria with the same prism, base out, before one eye. Or if one puts a prism of 10 prism diopters or more, base up or down, before one eye of the squinting patient he will see double images one being either exactly above the other, or showing in addition to the verti-

cal a very small lateral separation, at most one or two degrees; again about the same as would occur if persons with orthophoria were to localize the double images produced by the same prism. One may produce successively in either eye a horizontal and a vertical afterimage of a glowing filament the center of which has been fixated for about ten seconds while the other eye was covered. Or the two afterimages may be produced simultaneously by an adequate arrangement on the haploscope, so that each eye will see only one of the two glowing filaments the center of which is imaged on the fovea. If the positive or the negative afterimages form a cross, the existence of normal retinal correspondence based on innate conditions is evidenced. The cross will be seen unaltered if one eye is brought passively into a squinting position; for instance, by displacing it with the finger or with forceps. The same is true in cases of paralytic deviation, but only in a minority of cases with concomitant squint; namely, those in which the retinal correspondence is undisturbed. If the same afterimage test is made on a patient belonging to the aforementioned group of convergent squints, where the double images do not show the relationship which would be expected in cases with normal retinal correspondence according to the disparate position of the retinal images of an outlying object, the great majority of the patients will see instead of a cross the vertical afterimage produced in their right eye shifted more or less to the left, either crossing the left half of the hori-



Figs. 16, 16a, 16b (Bielschowsky). Schematic illustration of normal and anomalous retinal correspondence in convergent strabismus. Right eye turned in (visual line f_2C). F is formed on the left fovea (f_1) and on eccentric point (a_2) of the right retina. In a case with normal correspondence the afterimages of a horizontal and a vertical line produced in the left and right eye in succession would be seen forming a cross (fig. 16a) because the centers of the two retinae (f_1 and f_2) bear the centers of the horizontal and vertical afterimages, respectively, and localize them in one and the same—namely, the principal—visual direction (FF). If the same patient had an anomalous retinal correspondence adapted to the angle of squint, the afterimages produced in the same way would appear separated from each other as illustrated in figure 16b, the centers of the afterimages lying approximately in the direction of the two visual lines f_1F and f_2C. In figure 16 the straight lines connect the corresponding points of the two retinae in a person with normal correspondence, the dotted lines those in one with anomalous correspondence adapted to the angle of squint.

zontal image or passing beyond the left end of it (fig. 16). If the distance between the patient's eye and the glowing filament were 50 cm. while the afterimages were produced, their length would correspond to an angle of approximately 22 degrees, so that from the patient's statement as to the distance between the centers of the afterimages one can easily and rather accurately calculate the angle at which the visual directions of the foveae and of each other pair of corresponding points deviate from one another. Thus, in the cases under discussion, the foveal images of the two eyes are not localized as in cases with normal correspondence in one and the same—namely, the principal—visual direction, but in different directions, approximately coinciding with the geometrical (foveal) lines of direction. In other words, the image of the fixated object, formed on an eccentric point of the squinting eye is localized approximately at the same place where the foveal image is seen by the fixating eye. The same conditions, *mutatis mutandis*, are to be found in cases of divergent squint with an anomalous retinal correspondence. The image of the fixated object formed on an eccentric point in the temporal periphery of the right retina will be localized in the same visual direction as the left foveal image; accordingly, the center of the vertical afterimage produced in the right eye will be displaced to the right, either crossing the right half of the horizontal afterimage produced in the left eye or passing beyond the right end of it (fig. 18). If the two afterimages are produced in such a way that the center of the horizontal afterimage is formed on the fovea of one eye while the center of the vertical afterimage is imaged on the eccentric point of the other eye on which the objects fixated by the former eye are imaged, the vertical afterimage will pass exactly or approximately through the center of the horizontal afterimage. In these cases anomalous correspondence displays a functional adaptation of the sensorial apparatus to the anomalous position of the eyes relative to the fixated object.

In the face of this adaptation one would expect binocular single vision notwith-

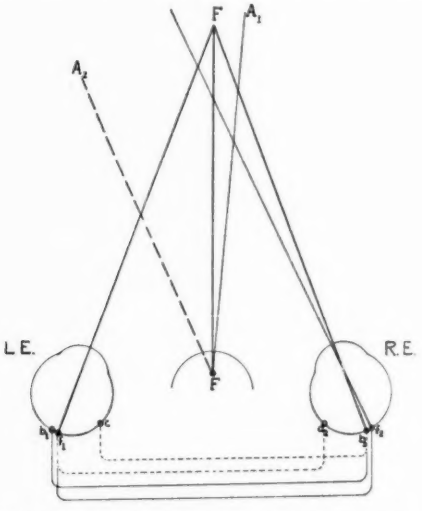
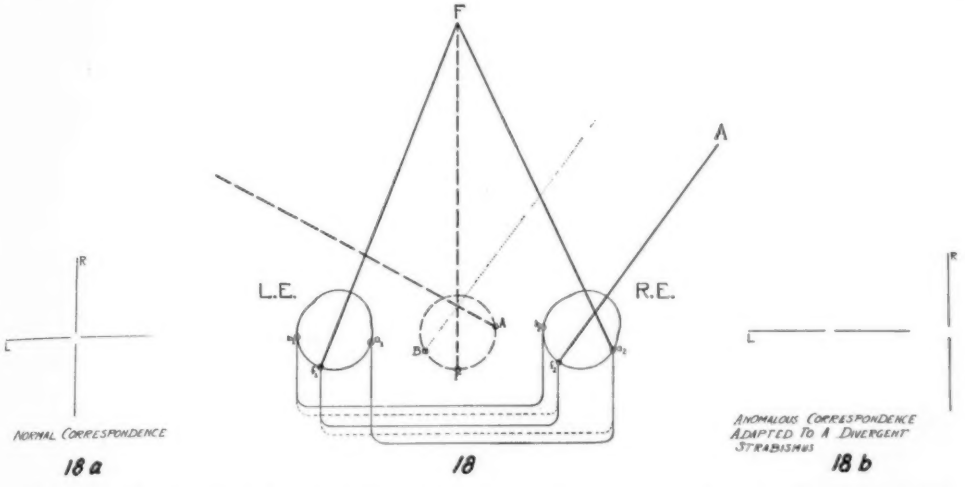


Fig. 17 (Bielschowsky). Rivalry between normal and anomalous correspondence arising in convergent strabismus with anomalous correspondence, as illustrated in figure 16 after the angle of squint has been reduced from 30 degrees to 5 degrees. The fixated object (F) will be seen at one time in crossed, at another in uncrossed double images; sometimes even triplopia may occur, the crossed image appearing simultaneously with the uncrossed one, beside the image seen with the fixating (left) eye. Crossed double images will be seen as long as the visual directions are determined by the anomalous correspondence according to which the images formed on a_2, as well as on f_1 were localized in one and the same visual direction (FF). An image formed on b_2 must be localized in the direction of A_2 to the left of F, since b_2 lies to the right of a_2. Uncrossed double images, the distance of which coincides with the angle of squint (convergence of 5 degrees) point to normal correspondence, since the image formed on b_2 must appear in the direction of A_1 to the right of the median plane in which both the foveal images are localized. In exceptional cases three images are seen: one, belonging to the left eye, in the direction of FF between the two other images belonging to the right eye.

standing the squint. But the majority do not even show rudiments of binocular cooperation. In the investigation of many hundreds of patients with strabismus I found only rudiments of binocular vision, and these in only a very few cases. A light or white disc appeared colored to such patients if a colored glass was put in front of the squinting eye. If simple drawings were presented to them in a stereoscope they were able to see the drawings combined or superimposed but without depth perception and without the phenomenon of physiological rivalry which will be found, as a rule, in normal binocular vision if pictures of different kinds and colors are presented in a stereoscope. I found a certain amount of depth perception only exceptionally in cases of strabismus. If Hering's drop test was arranged in such a way that the images of the dropping balls were seen with peripheral retinal parts of approximately equal acuity, a few patients were able to judge the distance of the dropping balls relative to the point of fixation obviously better than they did if the squinting eye was covered during the test (fig. 19). Fusion movements could never be produced in a case of squint. Most of the patients did not even show the aforementioned rudiments of binocular coöperation. The deficiency is due partly to the inadequacy of the eccentric retinal image of the fixating eye, partly to the inaccuracy and unsteadiness of the anomalous correspondence in which not one point of one retina corresponds exactly to one point of the other retina, but to an area of relatively large extent. Apart from that, it is obvious that a function acquired during life cannot be more than an imperfect substitute for a function based on a congenital foundation. But the main reason why in most cases of squint there is not even imperfect binocular vision is given by the fact that the images of the squinting eye in patients with



Figs. 18a, 18, 18b (Bielschowsky). Normal and anomalous correspondence in divergent strabismus. The two afterimages form a cross (fig. 18a) if the two foveae localize the centers of the afterimages in one and the same visual direction (normal correspondence). The afterimages appear separated (fig. 18b), the vertical one belonging to the right eye shifted to the right, if the relationship between the two retinae has been adapted to the divergent position of the visual lines. The straight lines (fig. 18) connect corresponding retinal points in normal, the dotted lines those in anomalous correspondence.

anomalous correspondence, as a rule, are suppressed just as they are in patients who have developed no anomalous correspondence. If a light is moved across the patient's visual field while he is looking straight ahead with glasses of different colors before the two eyes, the light will show the color of the glass that is in front of the fixating eye except in a small part of the visual field bordering the squinting visual line, where the light will show the color produced by the glass that is in front of the squinting eye. There is no mixing of colors in any part of the visual field.

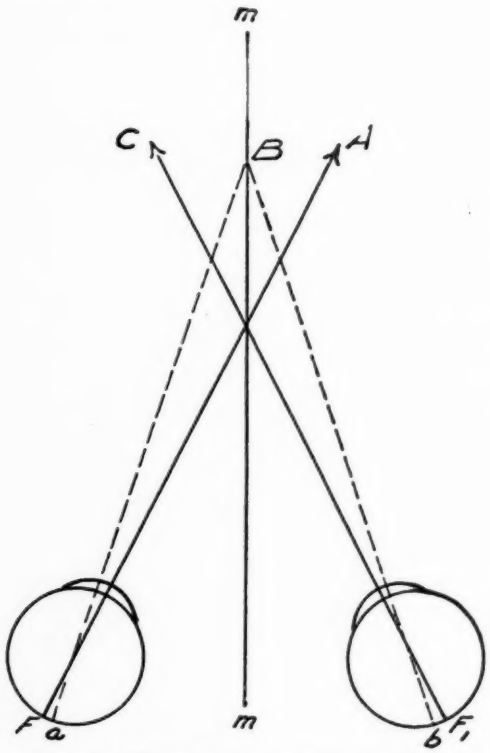


Fig. 19 (Bielschowsky). Hering's drop test modified for testing binocular depth perception in cases of convergent squint. Instead of looking at B, located in the median plane (mm) the patient has to fixate with his left eye the point A. If the distance between A and B subtends an angle of about half the amount of the angle of squint, the squinting right eye will be in such a position that the angle formed by F_1 C and b B will be equal to the angle between FA and aB, and the ball dropping very near to B will be imaged on retinal points of approximately equal eccentricity.

The conditions are not yet known upon which depend the acquisition of an anomalous correspondence during the existence of the squint. Investigators agree only on one fact; namely, that as a rule no anomalous retinal correspondence is to be found in squint arising after the sixth year. There are exceptions to this rule. Some patients with a high exophoria that becomes a manifest divergent squint temporarily, possess perfect binocular vision as long as the exophoria is kept latent by the fusion apparatus. But they do not see double images during the periods in which they show a manifest squint. When double images are produced by means of prisms combined with a dark-red glass they present the characteristics of anomalous retinal correspondence adapted to the angle of squint. On the other hand, there are patients who have had a permanent strabismus since the second or third year of life, in whom double images as well as afterimages show the characteristics of an intact normal retinal correspondence. In other cases where, in all the tests, only anomalous correspondence could be ascertained, after an operative correction of the deviation, sometimes immediately and spontaneously, the highest degree of binocular vision may take place. This is proof that the normal sensorial relationship between the two eyes may already have been established in earliest childhood without manifesting itself during the period of squinting. Now, considering the fact that the retinal images of the squinting eye are suppressed regardless of whether or not the features of an anomalous correspondence are present, the practical purpose of taking the trouble to perform all the time-consuming examinations concerning retinal correspondence, might be questioned.

CHEMICAL PATHOGENESIS OF CATARACT*

ARLINGTON C. KRAUSE, M.D.
Chicago

Cataract may be defined as a lens with an opacity resulting from the response of the lens to injury. The term, injury, is used here in the broad sense. This definition is based on the commonly accepted conception in general pathology that disease represents the response of the tissue to injury.[1, 2] No specific etiologic agent is the universal basic cause of all cataracts, for injury to the lens may arise from innumerable causes, such as anomalous developmental malformations, trauma, radiations, toxic substances, local ocular diseases, infections, metabolic deficiencies, and systemic diseases. Thus the cataract may result from immediate reactions on the lens or from remote reactions on tissues of the body. The response varies with the nature and severity of the injury, the particular lenticular structure involved, the duration of the reaction, and the age of the lens. The general pathological process of cataractous formation, however, is essentially based on the reaction of the lenticular cell to these injuries, which also may be logically classified as lenticular, ocular, and systemic, although the latter two types of injury are essentially a part of the first.

In clinical ophthalmology, cataract may be considered as the altered physiological function of the lens by nature's experimental methods. Since altered physiology is pathology and is ultimately associated with chemical processes, cataract may be regarded as the result of altered chemical processes that occur in the lens. At the present time there has accumulated a sufficient number of facts relating to the normal chemistry of the lens and other tissues to allow the formulation of a general chemical pathology of the lens.

* From the Division of Ophthalmology, Department of Surgery, University of Chicago.

THE NORMAL CHEMICAL PHYSIOLOGY OF THE LENS

There is reason to believe that the chemical physiology of the lens is similar to that of other body tissues. During evolutionary development of the organism the general plan of tissues is modified structurally and chemically to form a physical lens in the visual apparatus so that the animal, man, may better adapt himself to his environment. The lens has primarily a vital survival value only and occurs in many forms in other organisms, generally for receiving light and rarely for projecting light.[3] The lens is transparent and optically denser than the adjacent fluid and tissues so that it may serve its purpose. Its tranparency is probably not a retention of the translucency of embryonal tissues, as is commonly asserted, for innumerable adult transparent tissues and organs occur in nature for which there is no optical function.

THE CHEMICAL COMPOSITION OF THE LENS

The first step in the study of the normal clinical physiology of the lens is the analysis of the various compounds which form and take part in the reactions occurring within it. If a rough approximation may be made of the number and kinds of lenses used in analytical work in the past, one may give estimates of 500 rabbit lenses, 1,000 human lenses (chiefly for weight determinations, the remainder used for chemical analysis by Salit), and 120,000 bovine lenses (110,000 by the

TABLE 1

APPROXIMATE CHEMICAL COMPOSITION OF THE WHOLE LENS* IN PERCENT OF WET WEIGHT OF ONE-YEAR-OLD BOVINE LENS

Water	67
Protein	
Capsule	0.33
Albuminoid	3.7
Alpha crystallin	9.6
Beta crystallin	15.9
Albumin	0.45
Peptone	0.06
Nucleoprotein (as nucleic acid)	0.02
Mucoprotein	0.25
Lipids	
Lecithins	0.12
Kephalins	0.03
Sphingomyelins	0.04
Cerebrosides	0.004
Cholesterol	0.07
Fats	0.004
"Carotenoids"	0.0002
Free fatty acids	0.002
Other unsaponifiable substances	0.05
Water-Soluble Extractives	
Carosine	0.0047
Spermine	0.0043
Creatine	0.028
Creatinine	0.004
Inositol	0.150
Citric acid	0.002
Malic acid	0.01
Urea[39]	0.021
Glutathione[40]	0.220
Ascorbic acid[40]	0.026
Glucose	0.145
Potassium[41]	0.404
Sodium[41]	0.046
Calcium[41]	0.006
Magnesium[41]	0.008
Chloride[41]	0.069
Phosphate[41]	0.043
Lactoflavin	present
Fumaric acid	present
Succinic acid	present
Creatine phosphate	present
Guanidines	present
Ammonia	present
Amino acids	present
Pyruvic acid	present
Dihydroxyacetone	present
Lactic acid	present
Acetic acid	present
Formic acid	present
Oxalic acid	probably present
Ethyl alcohol	probably present
Oxaloacetic acid	probably present
Uric acid	probably present
Adenosine phosphate	probably present
Inosinic acid	present
Cytochrome	not found
Anserine	not found
Spermidine	not found
Iron[15]	not found
Copper[15]	not found

writer). It is easily seen that bovine lenses have been the most convenient source of fresh material in large quantities for analytical purposes. No one asserts that the metabolic processes are quantitatively identical in the human and in the bovine lens or in any other kind of lens; but there is reason to believe that the reactions of the human lens are of the same order, although, perhaps, not of the same degree, as those of other mammalian lenses. With the present modern procedures many kinds of analyses cannot be made on single human lenses, although they are the lenses of choice. Moreover fresh, whole, normal human lenses are never obtainable in large quantities at one time.

For convenience the lens may be considered as composed of three chief fractions; namely, proteins, lipids, and extractives (see table 1). The division into these fractions is chiefly based on the general grouping of technical procedures used in the analysis of tissues.

Proteins. The lens is composed of an unusual amount of protein, the detailed metabolism of which need not be given here.[4] In the first stage of growth the lens fiber is mainly made of albumin (gamma crystallin of Woods), alpha, and beta crystallin.[5] As the lens fiber ages the alpha crystallin is slowly changed into insoluble albuminoid, while beta crystallin remains practically constant in amount.[6] The mucoprotein, which may be the cementing substance of the fibers and may be in part composed of substances that make the protein of the capsule, has little to do with the other proteins within the lens fiber. Stimulation of the epithelial cells, however, may increase the formation of the hyaline capsular protein.

As may be seen by the table of analysis

* Data obtained by the writer are used unless reference number is given.

of lenticular proteins, the proteins are composed of the common amino acids (table 2). The analyses are qualitatively and quantitatively incomplete and other amino acids may be discovered later.

For the synthesis of the lenticular proteins, amino acids enter the cells after passing through the capsule from the aqueous humor. Whether or not the protein is formed from conjugation of simple chains of amino acids or from the addition of single amino acids to build a complex polypeptide, called protein, is unknown. It seems as if the simpler lenticular proteins, such as proteoses and peptones, only help to hold the more complex proteins in solution and are not merely precursors of proteins of higher molecular weight, for they persist in relatively fair quantities in mature fibers.

Lipids. The lipids in the lens are similar to those found in other tissues and take part in vital processes[7] (table 1). Their function is relatively unknown. By analogy there is reason to believe that cholesterol arises within the lens, for it is formed and oxidized by other tissues. The fats, perhaps, have no energy value, for the lens derives its energy chiefly from carbohydrates and its growth from amino acids. The more complex lipids, such as lecithins and kephalins, are in part necessary for the life of the cell.

Water-soluble extractives. The more active metabolic substances are in solution and they fall particularly under the title of water-soluble extractives (fig. 1). The most important metabolic process in the lens is that which involves the carbohydrate, glucose.[8] The energy derived from the breakdown of glucose is used for maintenance of cells at and above a vital basic equilibrium for the growth of the epithelial cells and fibers, and for the formation of the capsule.

Most of the energy is derived from the process which forms lactic acid from glucose (fig. 1). This process evidently occurs in cyclic equilibria. One of the high points of metabolic activity seems to deal with adenosine triphosphate, which is derived from adenylic acid by the exchange of phosphates from creatine phosphate, which, in turn, acquires phosphate from active hexose phosphate. A part of the hexose phosphate is split into two carbohydrates containing three carbon atoms which react and rearrange molecularly to form lactic acid. Certain ferments and coferments are necessary to bring about these changes, which require no oxygen but which give up energy for other vital processes, such as growth and maintenance of vital equilibrium. That the chief carbohydrate metabolism is the breakdown of glucose to lactic acid is indicated by the seeming lack of cytochrome in the lens.[9] Apparently the lens contains no iron respiratory enzyme of Keilin[10]—that is, cytochrome—and no enzyme (Atmungsferment) of Warburg.[11] If this is true it differs in this respect from the rest of tissues in the eye and body with possible exception of the cornea. This apparent absence of cytochrome may explain the fact that the lactic acid

TABLE 2

AMINO ACIDS OF THE LENTICULAR PROTEINS[42]

Amino Acid	Capsule	Albuminoid Percent	Alpha Crystallin	Beta Crystallin
Glycine	—	0	0	0
Alanine	—	0.8	3.6	2.6
Valine	—	0.2	0.9	2.1
Lencine and isomeres	—	5.3	5.7	2.8
Aspartic acid	—	0.5	1.2	0.4
Glutamic acid	—	4.6	3.6	2.7
Tyrosine	5.00[5]	3.6	3.5	3.7
Proline	—	1.9	1.8	1.4
Phenylalanine	—	4.6	5.5	4.1
Lysine	—	3.8	3.8	4.6
Histidine	—	2.7	3.8	3.6
Arginine	—	10.3	8.0	7.5
Cystine	1.48[5]	3.1	2.3	4.9
Serine	—	+	+	+
Tryptophane	0.58[5]	0.71[5]	0.85[5]	1.37[5]
Methionine	—	0[9]	0[9]	0[9]

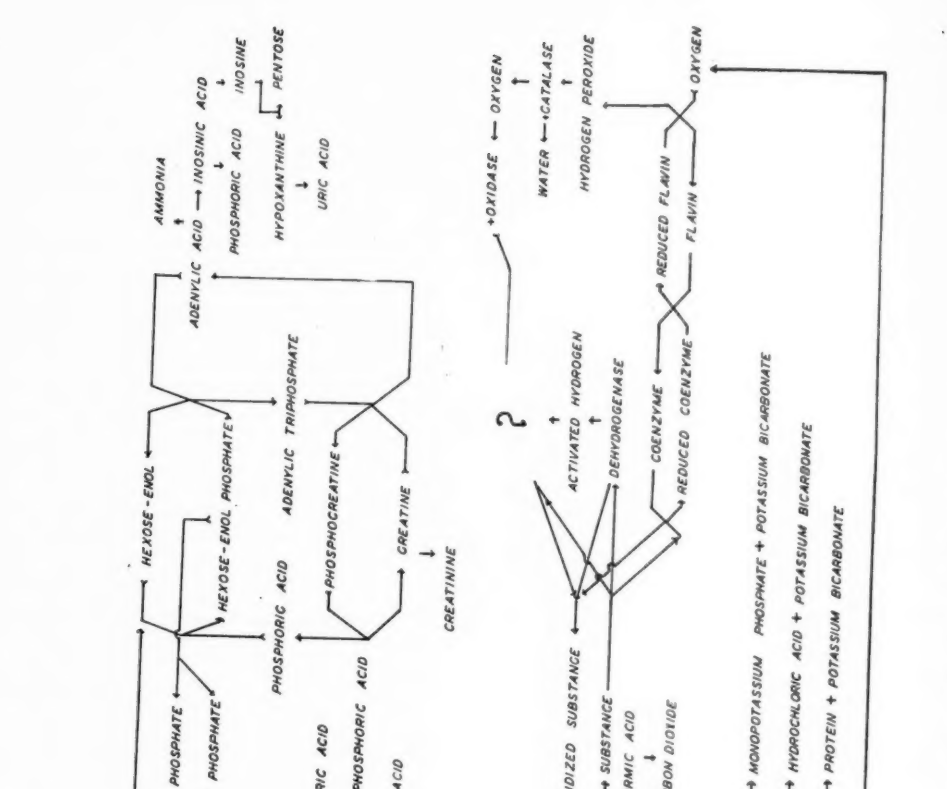


Fig. 1 (Krause). A hypothetical scheme of the carbohydrate metabolism of the lens (modified from Krause[5]).

becomes decreased in the aqueous humor in aphakia.

A smaller part of the energy of the lens evidently comes from the oxidation of lactic acid to carbon dioxide and water (fig. 1). In contrast to the mechanism which converts glucose to lactic acid, this process requires oxygen. Lactic acid undergoes many direct and cyclic reactions. Its derivatives are changed into other substances by gain or loss of hydrogen, oxygen, and molecular water and by other complex chemical reactions. Some of the products formed from lactic acid in these reactions are pyruvic, malic, succinic, fumaric, formic, and oxaloacetic acids.[9] Since oxygen in solution does not oxidize these substances, enzymes are necessary. In aerobic oxidation, which occurs in practically all mammalian tissue, cytochrome and indophenol oxidase play an important part. Since these substances have not been found in the lens so far, this type of oxidation may not occur in the lens. The lens evidently has an unknown dehydrogenase system and has another system which apparently is related to flavin, or vitamin G, and the enzyme, adenyl nicotinicamide phosphate. Both of these systems seem to cause enzymatic oxidations in the lens. The part glutathione plays in lenticular, much less in tissue, oxidation is doubtful. According to Hopkins it may act as a hydrogen acceptor in oxidations and may also effect the conversion of methylglyoxal to lactic acid.

The significance of other organic substances in cellular activity is in doubt. Carnosine[12] and inositol[9] occur in relatively large quantities and supposedly have an important function. Ascorbic acid is seemingly unnecessary for lenticular vitality.[13] Creatinine is concerned in the metabolism of creatine and creatine phosphate. Citric acid, methylguanidine, oxalic acid, and ethyl alcohol are substances which as yet have no place in the vital scheme. Amino acids are needed to make proteins, and also the peptides—carnosine and glutathione—and amino-acid derivatives such as spermine.[14] Not much is known concerning the actual role these substances play in the lenticular processes with the possible exception of glutathione. Much investigation will be necessary before these substances may be fitted into the metabolic mosaic of the lens.

The inorganic substances have many functions in the lenticular economy. The basic and acidic ions give a protoplasmic equilibrium. Calcium evidently hinders the solution of the protein after an open-wound injury of the lens. Magnesium takes a necessary part as a co-enzyme in the exchange of phosphate in glycolysis. Potassium enters into the intracellular reactions occurring within the protoplasm and nucleus. Chloride is the common inorganic anion which helps in the maintenance of the acid-base equilibrium and the distribution of water. Carbon dioxide is derived from oxidation of lactic acid and perhaps other substances. No iron or copper has been found in the lens.[15]

Growth. A brief description of the normal growth of the lens is necessary to the understanding, later, of the discussion of the chemical pathology of cataract, for during growth chemical changes take place which influence the physical structure and play a part in the physiology of the lens.[16]

The formation of the lens begins with the proliferation of the ectodermal epithelial cells over the primary optic vesicle to form the lens plate (fig. 2—1). From the invagination of this plate (fig. 2—2, 3) arises the lens vesicle (fig. 2—4). The posterior cells lengthen and fill the vesicle (fig. 2—5, 6). The peripheral epithelial cells form the capsule. During life the equatorial cells of the anterior epithelium elongate and form concentrate layers

Fig. 1 (Krause). A hypothetical scheme of the carbohydrate metabolism of the lens (modified from Krause[9]).

Fig. 2 (Krause). The growth of the lens. The form and time but not size of the lens are relative. 1. Primary optic vesicle (20 days old). 2. Beginning invagination of lens plate (25 days). 3. Lens vesicle being pinched off (27 days). 4. Lens vesicle (32 days). 5. Proliferation of posterior epithelial cells of lens (35 days). 6. Closure of lens vesicle (37 days). 7. Formation of peripheral fibers (52 days). 8. Lens acquiring lenticular shape (90 days). 9. Lens at birth. 10. Lens at 18 years. 11. Lens at 40 years.

around the first posterior cells, filling the vesicle (fig. 2—7 to 11). The older layers become more central as the younger layers are laid down peripherally.

The metabolism of the lens is not at a constant level throughout life because the lens grows and later becomes senile, like other tissues in the body of man and higher animals. The metabolism varies with the rate of growth and therefore is greatest with the formation of the lens and decreases during life as the lens grows.[17] In the early period of growth, water, the medium in which the substances react, is present in relatively great amount.[18] The water-soluble substances such as organic phosphates[19] and metabolic acids are in high concentration. As time passes and the metabolic activity decreases these metabolic substances also decrease in amount. This fact is recorded in innumerable reports. Moreover along with the aging process the soluble alpha crystallin turns into insoluble albuminoid.[6] This change of protein with age is comparatively slow in proportion to the rapid growth in the beginning and is faster in the adult lens. For example the subcapsular cortex contains little or no albuminoid until the lens practically stops growing, but, in contrast, the senile nucleus is mostly albuminoid.[9] This fact is shown by the following evidence: The ratio of insoluble albuminoid to soluble protein in the whole lens is 6:26 at 5 weeks, 15:19 at 4 years, and 21:15 at twelve years.[17] Comparing six equal fractions of weight in passing from the cortex to the nucleus, the ratio of insoluble albuminoid to soluble alpha crystallin in one-year-old lenses is 0.5:99.5, 5:95, 7:93, 10:90, 13:87, and 39:12.[6] The ratio of beta crystallin to total protein is practically constant in the same respective weight-strata.[6] Thus as the fiber of the cortex in time becomes the fiber of the nucleus the soluble alpha crystallin turns into insoluble albuminoid. With increase of albuminoid the fiber tends to hold less water and becomes more optically dense. There is reason to believe that albuminoid micelles are dispersed in solution of other proteins and as they become large aggregations of micelles they become more visible.

Senescence. The total growth of the lens reaches asymptotically toward a maximum. It is regular except, perhaps, in periods of physiological stress, such as birth and puberty, for which there are indications in the finer structure of lens. In mammals the growth of the lens is definitely limited long before senescence begins.[16] In man it is prolonged in the period of youth, which extends beyond the corresponding physiological time of other mammals. Furthermore, in man as in other mammals, it is probable that the size of the lens varies with the nutrition and the racial stock. For these reasons it may appear that the human lens grows anomalously for an infinite time. There are no unquestionable published data on the relation of weight to the age of the human lens. It is possible that some ophthalmologist in a town with a uniform population, studying specially fresh nonpathological lenses, will be able to answer this question definitely.

Senescence begins after maturity.[20] It is observed in the lens as well as in other tissues of the body.[21, 22, 23, 24] In senility, the hair turns gray, the menopause sets in, the skin becomes atrophic, and so on. The lenticular process is similar to that of other tissues. There is evidence for the inheritance of longevity of the body, the organs, and the lens. In fact, Steinhaus[25] and Berstein[26] found that there is a relationship between degree of lenticular sclerosis and the biological age of the body. The processes of senescence may be rapid or slow, and, in contrast to the growing lens, the catabolic activity ex-

(27 days). 4. Lens vesicle (32 days). 5. Proliferation of lens plate (25 days). 3. Lens vesicle being pinched off lens vesicle (37 days). 7. Formation of peripheral cells of lens (35 days). 6. Closure of 9. Lens at birth. 10. Lens at 18 years. 11. Lens at 40 years. 8. Lens acquiring lenticular shape (60 days).

ceeds the anabolic. If senescence of the lens is sufficiently advanced the cells begin to die.

From the foregoing discussion one may understand that normally there are many dynamic cyclic and direct processes that are taking place simultaneously in the lens. Various enzymes and substances are involved in many equilibria in the aqueous media. Substances are formed and broken down. Some are eliminated and others are acquired by way of the flow of the aqueous humor through the capsule. As the growth approaches a maximum the metabolism reciprocally approaches a minimum. A derangement of metabolism may be caused by a deficiency of vital substances or by the accumulation of metabolites. It may be the result of the interference with, or acceleration of, one or more reactions in the lens by change of temperature, action of toxic substances, or any other means. Furthermore, an altered lenticular metabolism may be inherited in the same manner as other defective chemical conditions, for example, such as congenital tyrosinosis or cystinuria.[27] If the metabolic dysfunction is sufficiently severe or prolonged, an opacity may be the result.

TABLE 3

THE INITIAL CAUSES OF OPACITIES IN THE LENS

Form	Examples Type of Cataract	Initial Chemical Effect
Mechanical	Concussion	Precipitation of protein
	Contusion	Necrosis of cells
	Traumatic	Increase of acid
Thermal	Heat workers	Increased enzyme action
Radiational	Radium	Destruction of enzymes
	Roentgen ray	Loss of electric charge of particulate matter
Chemical	Toxic	
	naphthalene	Cystine removed
	dinitrophenol	Creatine and adenosine, phosphate affected
	Vitamin G	Lack of flavin, necessary for growth
	Myotonic	Interference with creatine metabolism
	Diabetic	Increase organic acids
		Glucose metabolism affected
	Complicated uveal vascular stasis, intraocular inflammation, decreased uveal circulation, decreased permeability capsule by membrane around lens	Increased acid, decreased anabolism, increased catabolism, necrosis

THE CHEMICAL PATHOLOGY OF THE LENS

The alteration of the normal chemical physiology of the lens leads to chemical pathology. The pathological process resulting from the response of the lens to injury and ending with the formation of cataract is essentially the same whether it occurs naturally in man or experimentally in mammals, for the lenticular physiology of vertebrates is on the same general plan modified only by the peculiar physiology of the species.

In certain early conditions the minute structures of the lens may remain transparent but they may diffuse light by their increased optical density as compared with the surrounding media and give an apparent effect of cloudiness or opacity. This phenomenon is occasionally noticed in bundles of mature lenticular fibers as a precursor to the appearance of incipient cataract, particularly in the adult. Clinically this effect in the lens may be called a cataract if it interferes with vision.

Initial injury. The specific kinds of in-

jury are innumerable but they may be classified according to the nature of the injury; namely, mechanical, thermal, radiational, and chemical. The kinds of injury are listed in table 3.

Examples of the results of mechanical injury are concussion, contusion, and traumatic cataract. Concussion cataract apparently arises from the physical aggregation of protein micelles in the fibers through mechanical agitation.[28] If the protein aggregates are dispersed again, translucency returns. If there is no dispersion and the medium remains alkaline, no autolysis occurs, for, although the proteolytic enzymes are present, they are inactive in an alkaline medium. Trauma affects the lens in many ways, which will be discussed later.

Thermal cataract in man is the result of the action of infrared radiations and may be regarded as a radiational cataract. In man it is probably not merely the result of destruction of proteins by heat. There is good reason to believe that the equilibria are displaced and some enzymatic reactions are accelerated and others depressed by heat.

In radiational injury, if the effect on metabolic processes of other tissues may be applied to the lens the supposition may be held that the electrical charges of the particulate matter are changed, and the enzymatic mechanism is deranged.[29] The latter disturbance may explain the delay in the visibility of cataractous changes in radium and roentgen-ray cataract.

Under chemical injury there are a host of causes of cataract, such as interference with nutrition, deficiency of vital substances, and toxic disturbances of metabolism.

An interference with the general lenticular nutrition may be an obvious cause of injury. If the capsule is made impermeable by experimental methods, by an inflammatory membrane arising from exudation, or by any other means, the acidity is increased, and the normal lenticular metabolism is depressed. If it is arrested the lens dies and cataract results. A similar effect is produced directly by stasis of the aqueous humor or indirectly by stasis of the blood that supplies nutrition to the lens and also by anoxemia.[30] Cataracts which frequently arise from ocular inflammatory reactions are typical examples of this type of injury.

An interference with the general metabolism by the lack of substance necessary for growth or survival of the lens is another type of chemical injury. Since the body is unable to synthesize certain amino acids,[34] such as histidine, lysine, tryptophane, and several other amino acids, and must obtain them from the food, it is possible that the lack of these amino acids for building the peptides and the structural lenticular proteins may produce cataract particularly in growing animals. At least two factors may enter into this kind of deficiency. One factor is that, although the lenticular protein may have specific antigenic properties due to a certain group of coupled amino acids in the molecule which may be called ancestral protein or urprotein of the lens, the amount of the other amino acids in the protein may vary with the species. The deficiency of one kind of amino acid may therefore affect different species in various ways. The other factor is that species of animals vary in the use of amino acids in their vital economy and if necessary may take certain amino acids for other purposes than that for building protein. For example, some animals use glycine or glycuronic acid and others use cystine to remove toxic substances from the body. If it is necessary, available amino acids in the body may be preferentially used in this manner, even if they are ingested in the food. For instance, the detoxification of naphthalene and cholic acid, which

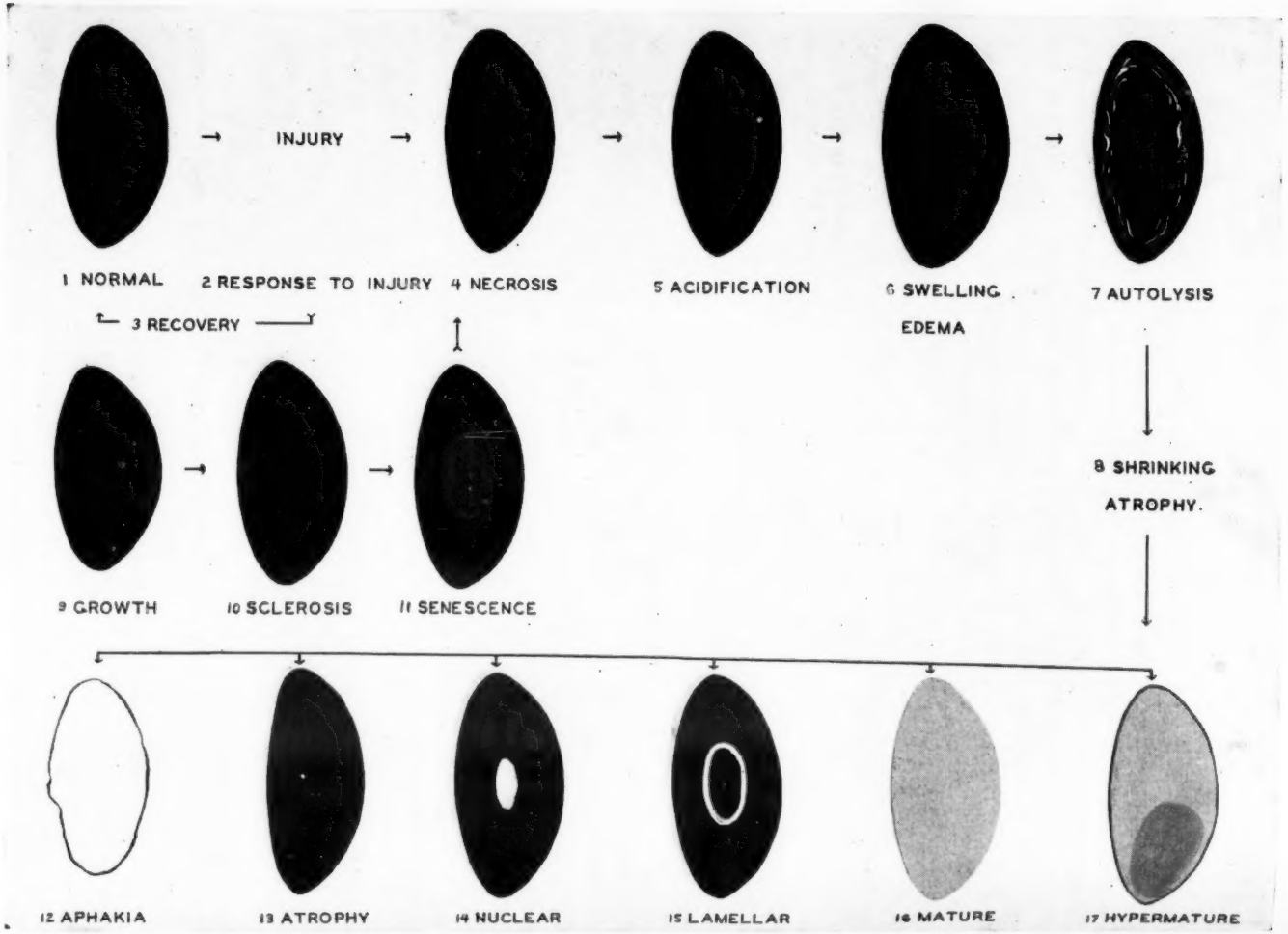


Fig. 3 (Krause). A diagram of the general chemical processes resulting in the common forms of cataract.

occurs by conjugation with cystine in rabbits, affects the lens apparently by the loss of cystine in the glutathione of the tissues.[31] In this case the injury by naphthalene is less marked with the ingestion of cystine and calcium, or with plant food containing these two substances.[32] Other metabolic deficiencies also affect the lens. Apparently in rats the lack of tryptophane causes cataract.[33] Cataract arising from the deficiency of vitamin B_2 or lactoflavin in the growing animals belongs to this class of nutritional deficiencies.[34]

Many toxic substances injure the lens directly or indirectly. Thallium poisoning in rats results in cataract, but in man the optic-nerve fibers are killed. This is a difference in the metabolism of species. If the reaction of the lens is comparable to other tissues, dinitrophenol cataract in man arises from the interference with the creatine and adenosine-phosphate processes in the lens.[35, 36] Other unknown factors influence the injury because relatively few people of the vast number taking this drug develop cataract. The lenticular injury resulting from the action of many other toxic substances need not be discussed, as the general effect is relatively known or more study will give the chronological story of the finer pathology.

Some types of injury seem to be an inborn anomaly of metabolism.[27] General examples are congenital cystinuria and tyrosinosis. Perhaps the cataract, which occurs in myotonic dystrophy in which the creatine metabolism appears to be defective, may be thus classified. Mongolian idiots also develop cataract and abnormally show phenylpyruvic acid in their urine.

It is not the visible final picture that is most obscure but the initial metabolic insult. An indeterminable amount of critical investigation is often necessary for the discovery of the primary effect.

Chemical pathology. The pathological chemistry of the general cataractous process starting the response to injury and leading to the relatively stationary state of cataract may be connected to general pathology and to the clinical picture of the lens (fig. 3).

After a slight injury, if no irreversible change takes place, recovery may be complete and no opacity or atrophy may occur (fig. 3—3). This effect may occur after a contusional cataract. In this case a peptization of the proteins probably occurs again.

A mild injury of long duration affecting one or more of the lenticular metabolic processes without producing necrosis may give rise to amorphous or crystalline opacities that are sometimes called precipitates or infiltrates when they are seen in other tissues. The formation of cholesterol esters is considered to be the sign of decreased metabolism and necrosis by pathologists. Calcium also tends to be precipitated as carbonate or phosphate under similar conditions. The protein crystals that occur in rare types of cataracts may be alpha crystallin, for this protein can be prepared in a pseudocrystalline form.[9] In other words, in a mildly altered metabolism, some normal metabolic substances are no longer in the proper concentration and may accumulate locally if they are insoluble or tend to form insoluble products.

Necrosis of a part or of the whole lens starts a series of chemical events beginning with acidification. The acids are chiefly lactic and phosphoric acids derived from glucose and organic phosphates. The acidification increases the number of ions and molecules, and therefore the osmosis is greater within the lens (fig. 3—5). Water passes through the capsule and the lens swells or is edematous (fig. 3—6). The increased acidity is now sufficient to cause the enzymatic breakdown of the proteins, or

Fig. 3 (Krause). A diagram of the general chemical processes resulting in the common forms of cataract.

17 HYPERMATURE
16 MATURE
15 LAMELLAR

autolysis, which gives more molecules and ions, and therefore more osmosis occurs (fig. 3—7). The lens is now an intumescent cataract. The soluble fragments of protein arising from proteolysis and the soluble extractives leave the lens; it shrinks or atrophies (fig. 3—8). The insoluble albuminoid micelles form visible aggregations and the lens becomes an immature or mature cataract, usually depending upon the degree of autolysis (fig. 3—16). The final condition greatly depends upon the insoluble residue within the capsule. This residue is chiefly insoluble albuminoid.

Depending upon the age of the lens and nature of the necrosis, the result may be atrophy, aphakia, or a light-colored opacity. For example, in the young, growing lens, a slight opening of the capsule accompanied by an injury to the fibers may end in a complete recovery of clarity in the atrophic tissue (fig. 3—13). The products from the necrotic cells act as a stimulus[37] and cause the anterior epithelial cells to proliferate and to form new capsular protein. Since the subcapsular fibers contain little or no albuminoid there is no opacity. The end result is a clear but flatter and smaller lens. After a sudden extreme injury with complete loss of epithelial cells, the young lens may become entirely absorbed and only the capsule remain (fig. 3—12). If the injury is moderate and occurs before birth, a nuclear cataract may be the result (fig. 3—14) and if it happens in the young lens after birth, a lamellar cataract is formed[38] (fig. 3—15). The same degree of injury, which in the young lens may induce the types of cataract that have been discussed previously, in the old lens results in a mature cataract as an ultimate picture. The residue in the adult type of cataract is the large amount of insoluble albuminoid which resists the action of enzymes. Necrosis of the subcapsular cells or fibers produces substances which stimulate the proliferation of the cells and the formation of hyaline capsular protein. In some instances the proliferation of epithelial cells may interfere with the metabolism of the circumvallated lenticular fibers and the cataractous process is accelerated. If the capsule is made impermeable by its thickening through the stimulation of the epithelial cells, the internal fluid mass is retained in a sac. The nucleus, which is mainly insoluble albuminoid, remains undigested by the proteolytic enzymes. The amino acids arising from the proteolysis of soluble protein are in solution, and leucine and tyrosine tend to crystallize out as they are relatively insoluble. This condition is called a hypermature cataract (fig. 3—17). Cataract resulting from the biological degeneration of senility passes through similar changes.

Although the initial appearance of lenticular opacity is in general dependent upon the specific type of injury, the general process of cataract formation follows the course of events as described.

950 East Fifty-Ninth Street.

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THE TREATMENT OF CONCOMITANT CONVERGENT STRABISMUS*

Results secured by atropine, occlusion, and correction of the refractive error

Elmer A. Vorisek, M.D.
Chicago

Much has been written of the cross-eyed child, and this condition continues to be of considerable cosmetic importance to the patients and to their parents. Many medical and surgical procedures have been advocated for the correction of this physical defect, and their champions are legion. Probably nothing has attracted more world-wide interest in this connection than the recently revived method of orthoptic training. We are living in a machine age, and many are the contrivances that have been developed and are being sold for the purpose of making crossed eyes straight and amblyopic eyes to see again.

The writer's interest in the cross-eyed child naturally developed from an association with large numbers of these unfortunate children at the Children's Memorial Hospital Out-Patient Clinic. It has been the policy of our attending clinicians to refract carefully and to prescribe properly fitting lenses for as many children as possible. Time has not been available for orthoptic training *per se,* and for some years our children have been made available to Dr. George Guibor to serve as a check against children with similar conditions who were being treated at Northwestern University, where active orthoptic training is given.

A study of the "Changes of the refraction in children with convergent strabismus" (Amer. Jour. Ophth., 1935, v. 18, Sept., pp. 820-826) was reported by the writer before this Society in 1935, and it has been continued to include a much larger series of cases. While abstracting the voluminous records, a note was made of the kind and degree of deviation found upon the occasion of the first and last examination. This explanation is given in order to acquaint the reader with the fact that the figures produced here have been obtained impartially in consecutive cases. All of these children have been refracted by the method of cylinder skiascopy, under atropine, at least twice; those with only one refraction have been excluded from this report. Cases with corneal opacities, ectopia lentis, optic atrophy, trauma, or any additional pathological conditions have also been discarded.

Many children with monocular convergent strabismus have amblyopia, or at least a decrease of visual acuity in the deviating eye. At our clinic we are extremely anxious to discover this situation at as early an age as possible, and we make a strenuous effort to do so. We use the Snellen illiterate "E" chart; frequently a child less than three years of age will respond to this method. While it is not always possible to obtain an absolutely accurate measure of the vision, it is most important to know if there is a difference in the vision of the two eyes. In the event of defective vision, the fixating eye is atropinized or covered, or both, for varying periods, and we have, on occasion, kept the fixating eye covered with a gauze bandage and adhesive for a month at a time.

In conjunction with this use of occlu-

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sion and atropinization of the fixating eye we have also been partial to the practice of prescribing full correction of the refractive errors, and the free use of a cycloplegic to relax the accommodative effort. Refractions tested under atropine cycloplegia have been repeated as frequently as was deemed advisable. Our patients have been urged to return periodically, and those who failed to report when a refraction was ordered were contacted by mail, at least twice, in an effort to have them return. It is only natural that some patients obtained a maximum of attention and some much less; but on the whole we have found coöperation satisfactory.

The measurement of the angle of squint was made by having the patient fixate at a distant point first. Then the fixating eye was covered, and the resultant movement of the converging eye, in millimeters, was multiplied by the factor, five degrees. This procedure was then repeated for the normal reading distance. If a difference between these two determinations existed, the average was used, and, if any question arose, the greater angle was always recorded. Fusion or stereopsis was not determined in all of the cases, and consequently these factors will not be considered.

Some of the children had had a varying degree of attention elsewhere before entering our clinic, and may have been wearing glasses. In that case, the determination of the squint angle was made with the child wearing the glasses. The final determination was likewise made with glasses worn, and the excessive convergence frequently observed, incident to the removal of the glasses, will not be considered. Children reaching the age of 13 years are no longer admitted to our clinic, and, consequently, all observations recorded were made on children under 13. Practically all of the refractions have been made by one clinician, and most of the observations have been made by another. Surgical treatment will not be considered at this time, nor will the results of such treatment be reflected in our findings. Likewise, cases of paralytic strabismus will be ignored.

TABLE 1

INCIDENCE IN MONOCULAR GROUP—183 PATIENTS

Not Over 4 Years			Over 4 Years		
Degrees	Patients	Percent	Degrees	Patients	Percent
20 or less	49	27	20 or less	83	45
Over 20	26	14	Over 20	25	14
Total	75	41	Total	108	59

TABLE 2

RESULTS IN MONOCULAR GROUP—183 PATIENTS

	Not Over 4 Years				Over 4 Years			
	20° or less (49)		Over 20° (26)		20° or less (83)		Over 20° (25)	
	Patients	Percent	Patients	Percent	Patients	Percent	Patients	Percent
Straight	24	49	6	23	61	74	6	24
Improved	8	16	10	38	10	12	13	52
No change	5	10	9	35	6	7	5	20
Worse	12	25	1	4	6	7	1	4

These explanations have been offered not in apology for criticisms, which may be in order, but rather to acquaint the reader with the procedures employed.

For this discussion the records of 245 patients have been studied. Of this number 183 (75 percent) had a monocular strabismus and 62 (25 percent) had an alternating convergent strabismus.

The children have been separated into four groups, based upon observations made at the time of the original visit to the clinic. They were divided first according to age: those up to and including the age of four, and those five years of age and over. Each of these two groups was again divided into those having a convergent angle of 20 degrees or less and those with a greater angle. Then at the time of the final observation the cases were grouped according to results: Eye straight, Improved, Not changed, or Worse.

Eye straight refers to the position of the visual axis; and a variation of even 5 degrees from the fixation point was not considered under this heading; a variation of less than 5 degrees was regarded as straight. In those patients who originally had 20 degrees of convergence or less, a 5-degree reduction in the squint angle was considered an improvement, but for patients with a higher degree of convergence, at least 10 degrees was demanded as an improvement. Otherwise, the cases were considered as unchanged. A similar method was used in classifying the squints which became worse. In patients who had a convergence of 20 degrees or less, a 5-degree increase was considered as an increase in the strabismus; but in those who had a higher degree of convergence, an increase of at least 10 degrees was required in classifying cases as worse.

Among the 183 children with a monocular strabismus, 75 were in the younger age group, and of these 49 had a convergence originally of 20 degrees or less. After treatment, 49 percent became straight, 16 percent showed improvement, 10 percent were unchanged, and 25 percent became worse. Twenty-six cases in the younger age group with over

TABLE 3

INCIDENCE IN ALTERNATING GROUP—62 CASES

Not Over 4 Years			Over 4 Years		
Degrees	Patients	Percent	Degrees	Patients	Percent
20 or less	8	13	20 or less	26	42
Over 20	12	19	Over 20	16	26
Total	20	32	Total	42	68

RESULTS IN ALTERNATING GROUP—62 CASES

	Not Over 4 Years				Over 4 Years			
	20° or less (8)		Over 20° (12)		20° or less (26)		Over 20° (16)	
	Patients	Percent	Patients	Percent	Patients	Percent	Patients	Percent
Straight	none		none		13	50	1	6
Improved	2	25	8	66	3	12	11	69
No change	3	37	2	17	4	15	4	25
Worse	3	37	2	17	6	23	none	

20 degrees of original convergence showed that 23 percent were straight, 38 percent improved, 35 percent unchanged, and only 4 percent worse.

The older age group of 108 cases included 83 with 20 degrees or less of squint. After treatment, results showed 74 percent classified as straight, 12 percent as improved, 7 percent with no change, and 7 percent as worse. Over 20 degrees of squint angle was represented by 25 patients, 24 percent of whom became straight, 52 percent improved, 20 percent were unchanged, and only 4 percent worse. Of the 62 children in this series, the younger age group is represented by 20 cases, 8 with 20 degrees or less of squint. None of these became straight, 25 percent showed improvement, 37 percent were unchanged, while 37 percent became worse. Of the 12 patients having over 20 degrees of original convergence, again none became straight but 66 percent showed improvement, 17 percent did not change, and 17 percent became worse.

The older age group included 42 cases. An original convergence of 20 degrees or less occurred in 26 cases, 50 percent of which became straight, 12 percent were improved, 15 percent were unchanged, and 23 percent became worse. Over 20 degrees of convergence was originally found in 16 cases. Only 6 percent became straight, 69 percent improved, 25 percent remained unchanged, and none became worse.

Summary

Monocular group

An analysis of the data here presented shows that 72 percent of our patients had a convergence of 20 degrees or less, and that 64 percent of these made a complete recovery. In contrast, only 25 percent of the children with more than 20 degrees of convergence showed a recovery; however, the percentage of improvement was quite high.

Fifty-nine percent of our patients were over four years of age when first seen, and 61 percent of these made a complete recovery, while only 40 percent of the children in the younger age group recovered.

When no change occurs in the angle of deviation, we must consider the results unsatisfactory. If these cases are added to those in which the condition became worse, we find 26 percent of them in the younger age group and 16 percent in the older age group.

Taking the whole group of 183 children into consideration, 52 percent made a complete recovery, 24 percent were improved, 14 percent showed no change, while 10 percent became worse.

Alternating group

A group of 62 cases is hardly sufficient for definite conclusions to be drawn, but they were fairly evenly divided as regards the degree of squint. A convergence of 20 degrees or less was present in 55 percent, and only 38 percent of these made a complete recovery. Only one child with over 20 degrees of squint angle recovered completely, but of the 28 children in this group 19 showed an improvement.

The older age group contained 68 percent of the children, 29 percent of whom made a complete recovery; but in the younger age group none recovered.

From the standpoint of results, 50 percent of the children in the lower age bracket showed unsatisfactory response, and, similarly, 33 percent of the older children met failure.

Of the total number of alternating squints, we found that 23 percent became straight, 39 percent improved, no change occurred in 20 percent of the cases, and 18 percent became worse.

Conclusions

In general, the possibilities of obtaining a recovery or an improvement are much better in the monocular group, while the treatment of the alternating group is definitely unsatisfactory.

The percentages of unsatisfactory results found in the group of children with alternating convergent strabismus are exactly double those found in the group with monocular strabismus.

I wish to thank Dr. Richard C. Gamble, attending ophthalmologist at the Children's Memorial Hospital, for his coöperation in the preparation of this paper.

104 South Michigan Avenue.

SARCOMA OF THE IRIS*

A case report

Harry D. Rosenbaum, M.D.
Saint Louis

Sarcoma of the iris is a rare form of malignancy of the eye. Various statistics have been compiled as to its frequency. Knapp[1] had one case in 65,000 admissions; Komato,[1] one in 35,000. Up to 1937, about 176 cases of primary sarcoma of the iris had been reported in the literature. Of the various types, there are those that tend to grow solidly, invading the ciliary body and sclera, and those that tend to circle the iris, producing a so-called ring tumor. Others are classified as melanotic if they contain pigment, and as leucosarcomata if there is no pigmentation, the former being more prevalent than the latter.

The growth always begins as a small, concentrated, nodular-formed tumor, chiefly in the anterior layer. In rare cases many nodules may be present. Though numerous writers are of the opinion that these malignancies arise from nevi located in the iris, this fact is not at all proved, according to Ginsberg.[2] The growth of a sarcoma of the iris is usually protracted over a long period, at times with slow progress (as long as 20 to 30 years), and with this rather slow growth goes the rareness of multinucleated cells and mitotic figures. However, after long periods of relatively slow growth, the tumor may suddenly increase in size.

In sarcoma of the iris, as in sarcoma of the choroid, the pigmented epithelium can proliferate into the tumor without participating in the malignant formation. This is exemplified in the case to be reported. The cell form is, as a rule, not simple; although pure spindle-cell sarcomata do occur, they are usually composed of polygonal, bipolar, and short spindle cells, or mixed round and polygonal cells.

Nothing definite has been noted as to the age incidence of these tumors. However, many of the cases reported were in relatively young or middle-aged adults: Zentmayer's[3] in a 10-year-old boy, Knapp's[1] in a 30-year-old man, Patterson's[4] in a 40-year-old man, Myer and Kubik's[5] in a 54-year-old man. Although most cases of tumors reported were in eyes of males, many occurred in the eyes of females.

Of all malignancies involving the eyeball, only sarcomata of the iris, along with tumors of the conjunctiva, if recognized early, may be cured by local excision, not necessitating removal of the

* From the Department of Ophthalmology, Oscar Johnson Institute of the Washington University School of Medicine.

eyeball. A broad iridectomy may suffice if the case is seen early enough; nevertheless, this should be followed by radiation therapy and careful watching over a long period of time.

Case report

Mrs. F. B., white, 26 years old, was first seen in the Washington University Out-Patient Department on June 19, 1936. She gave the history of having had a small yellowish-brown spot on the iris, located inferiorly just to the temporal side of the vertical meridian. As far as the patient remembered, this had been present all her life and had caused no discomfort of any kind. It was quite small, but the patient could not state the exact size except to say that it was "a little larger than the head of a pin." About three years before her entrance into the eye clinic, she noticed that the small lump had begun to increase in size. This enlargement progressed gradually, but steadily, until in January, 1936, the patient discovered that the mass projected slightly into the pupillary area. The projection appeared to be fixed at its base but would move rather freely in the pupillary zone. The patient, however, was still free from any pain and inflammation in or about the eye, except for an occasional very slight ache. During the six months prior to her admission, the growth seemed to have inincreased in size more rapidly. There was no history of any previous injury nor of inflammation.

Essentially negative, the patient's past history disclosed that she had been married nine years, that there were three children, the youngest of whom was three years old, and that there had been no stillbirths and no miscarriages. She had lost 28 lbs. of weight between August, 1935, and June, 1936.

Gastrointestinal. About March, 1936, the patient began to have some nausea after meals, with an occasional vomiting spell. She also had frequent attacks of severe abdominal pain starting in the left lower quadrant and radiating down into the groin. These attacks would last 15 to 20 minutes. Two days before admission to the hospital, the patient had had four attacks of pain in the right lower quadrant; each lasting about two hours and necessitating her going to bed. No vomiting nor nausea was associated with these last attacks.

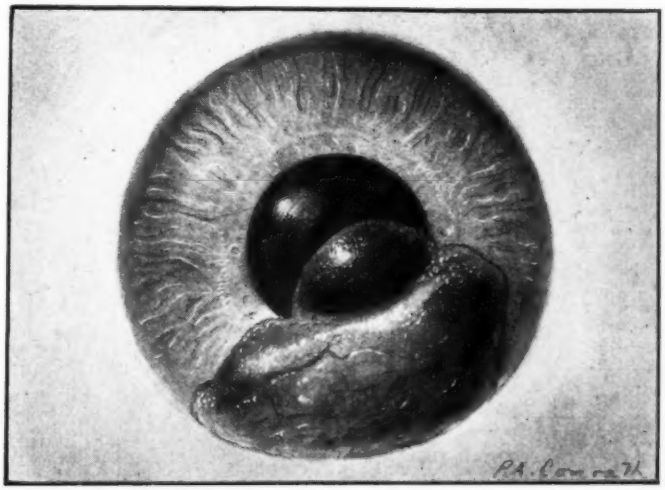

Fig. 1 (Rosenbaum). Drawing of anterior segment.

Examination

Vision of the right eye was 6/5, of the left 6/7.5. The lids and lacrimal apparatus were negative in both eyes; the conjunctivae and corneae clear.

Left eye. In the lower temporal quadrant was a large vascular yellow mass

growing from the iris and extending from about the 4- to the 7-o'clock position. This mass touched the posterior surface of the cornea at its highest portion. Above the mass was a small, deep brown, globular, cystlike projection formed of pigmented cells which filled about two thirds of the pupil. Shading off from the base of this to unaffected iris tissue was a strand of normal pigmented cells making an unusually wide ruff (fig. 1).

toms, surgical consultation was requested, and a diagnosis of chronic appendicitis was returned. The appendix was removed two weeks later. The pathological diagnosis was chronic appendicitis.

The patient was discharged from Barnes Hospital on June 24, 1936, and was followed further in the Out-Patient Department. When last heard from in March, 1938, the socket was doing well and she had no discomfort.

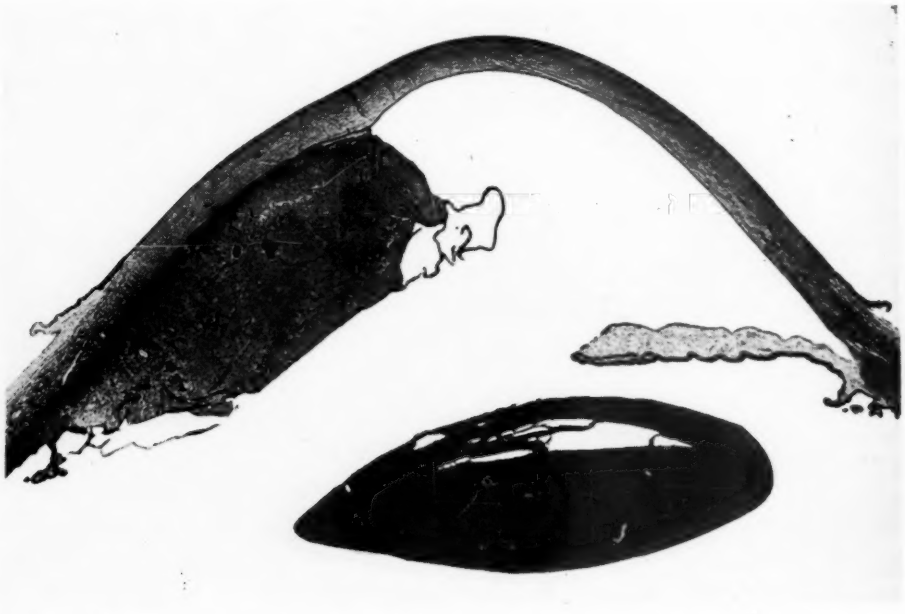

Fig. 2 (Rosenbaum). Section through the anterior portion of the eye, showing on one side the neoplasm replacing the iris (× 10).

The intraocular tension was normal; in the fundus no pathology was seen.

The patient entered Barnes Hospital on June 22, 1936.

Using local anesthesia, which consisted of 4-percent cocaine instillation, retrobulbar injection of 2-percent procaine hydrochloride, and Van Lint akinesia, an enucleation was performed by Dr. T. E. Sanders, and a glass ball implanted. The postoperative course was essentially uneventful.

In view of the gastrointestinal symp-

The eyeball was brought to Dr. H. D. Lamb, who returned the following pathological report:

Macroscopic findings: Vertical sections through the eyeball showed the iris on the inferior side to be replaced by a dense mass, solidly filling the anterior chamber of that side and, anteriorly, lying closely in contact with the posterior surface of the cornea (fig. 2). At the site of the former pupillary margin, the tumor-mass ended bluntly with its free anterior-superior margin lying at right angles to

the surface of the cornea. Posteriorly and inferiorly the neoplasm had invaded a little the anterior-internal angle of the ciliary body but not the ciliary processes. The anterior-superior and posterior-superior surfaces of the tumor mass did not meet in a solidly filled angle. Instead, a deep indentation into the neoplasm at this situation was filled by several large, clear, cystic spaces covered by pigmented epithelium of the iris. The more superior of these cystic spaces extended superiorly considerably beyond the anterior-superior surface of the tumor. It has been described in the clinical notes as filling about two thirds of the area of the pupil. At the position of the former base of the iris, on its posterior surface, also, several large clear cystic spaces were observed surrounded by pigmented epithelium of the iris.

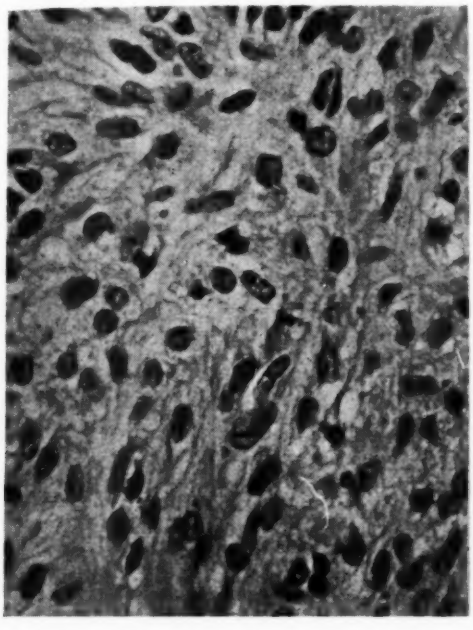

Fig. 3 (Rosenbaum). Section through the tumor, showing lack of uniformity of the constituent cells (× 450).

Fig. 4 (Rosenbaum). Section through the tumor, showing reticular fibers, Wilder's stain (× 275).

Microscopic findings: No part of the stroma of the iris remained on the inferior side, or where the neoplasm had formed. The posterior pigmented epithelium of the iris was present over the former pupillary zone and base of the iris. Between these two situations, in some areas, it had disappeared. In many places, but particularly near the former base of the iris, this posterior pigmented epithelium had proliferated for varying distances into the mass of the tumor.

The constituent cells of the neoplasm were not uniform, but were characterized by nuclei having spindle shapes of greatly varying length and thickness (fig. 3). The nuclei stained in a moderate degree and each contained a large nucleolus. The outlines of the cells could rarely be distinguished. The cytoplasm of the cells stained rather lightly.

When the reticular fibers were stained according to Wilder's method,[6] it was found that they were comparatively few in number (fig. 4), lying generally around

groups of tumor-cells.

The pigment melanin was but scantily present. Its arrangement, as always in uveal sarcoma, varied from isolated sarcoma cells stuffed with melanin to large groups of tumor cells sprinkled with fine grains of melanin.

Small capillaries were numerous in the neoplasm.

The cornea showed a flattening of the posterior endothelial layer from pressure by the tumor mass. Descemet's membrane was unaffected. The trabeculae of the filtration network were pressed together by the growth of the neoplasm and were infiltrated to a moderate degree with small lymphocytes.

The anterior-internal angle of the muscle-triangle of the ciliary body had been destroyed a little by the growth. The part of the tumor invading the anterior-internal angle of the ciliary body showed thicker reticular fibers than anywhere else in the neoplasm. The ciliary body posterior to the growth presented a moderately dense infiltration with small lymphocytes.

The remainder of the eyeball presented little of significance. The retina showed a secondary, diffuse, fine, clear cystic degeneration and the optic nerve a moderate degree of atrophy.

Comment

According to Callender and Wilder,[7] the tumors containing epithelioid, fascicular, and mixed-cell types are more malignant than those in which the predominating cell is the spindle-cell. This is borne out in this case; here the constituent cell was one containing nuclei, having spindle shapes of varying lengths and thicknesses. Another factor used to determine the relative malignancy of this type of growth is the presence and location of the argyrophil fibers. The above authors concluded that the presence of these fibers in all areas of the primary tumor is indicative of comparative benignancy; however, those that contained no fibers in the primary tumor were of the more malignant class. The reticular cells in the case reported were few in number, but were located around the tumor cells.

I wish to thank Dr. H. D. Lamb for his assistance in reporting this case.

640 South Kingshighway.

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BILATERAL ACCOMMODATION PARALYSIS AND UNILATERAL SCOTOMA IN SPHENOIDAL—SINUS DISEASE

A CASE REPORT

NORMA B. ELLES, M.D.
Houston, Texas

The unusual association of a bilateral paralysis of accommodation with a unilateral retrobulbar lesion seems worthy of recording, since no report in the literature of such a combination of symptoms has been found by the writer.

CASE REPORT

N. A., aged 20 years, a university student, was seen on June 13, 1933. While in Europe with a college band the previous summer, he had first begun to experience discomfort in the eyes with aching of the eyeballs and some dimness of vision. While in the university the past fall several attempts to fit glasses were made, but he was unable to wear any of them comfortably. To see reading matter he had to hold it very far off. The eyes were sensitive to sunlight.

The patient was very athletic. In January, 1932, while bob-sledding, he broke his pelvis, but this was unrecognized until his return home in summer when his physician had X-ray studies made and found the lesion, which had healed. He had had the diseases of childhood. Tonsils and adenoids had been removed when he was five years of age.

My records show that he had been given glasses when 9 years of age of the following correction: O.D. +1.50 D. sph. ⊂⊃ +0.50 D. cyl. ax. 90°, securing vision of 20/20; O.S. +1.50 D. sph. ⊂⊃ +0.50 D. cyl. ax. 90°, with vision of 20/20.

On June 16, 1933, the external examination was negative. The pupils reacted normally to light, but weakly to convergence. P.P., O.D. was 30 cm., O.S. 29 cm.

Vision uncorrected O.D. was 20/100, O.S. 20/40+. Retinoscopy under homatropine gave results as follows: O.D. +1.25 D. sph. ⊂⊃ +1.50 D. cyl. ax. 90°, vision = 20/20; O.S. +1.25 D. sph. ⊂⊃ +1.25 D. cyl. ax 90°, vision = 20/20. Glasses were prescribed accordingly with spheres cut 0.25. Slip-ons of +2.00 O.U. were given for near work.

Ophthalmoscopic examination was negative except that the maculae were slightly congested.

Blood Wassermann and Kahn reactions, the spinal-fluid Wassermann as well as the general physical examination, including the neurological examination were negative. The rhinological examination showed a slight deviation of the septum to the right, otherwise it was negative.

On July 18, 1933, the patient complained of seeing a large dark spot before the right eye. The right field, taken at this time on the perimeter with a 10-mm. white target, showed an irregular ring scotoma about 10 degrees in width, incomplete above. The left field was normal. Two days later the right field showed a complete ring scotoma slightly wider to the temporal side. The central area was clear.

On July 27, 1933, an X-ray examination of the sinuses was made by Dr. McDeed who reported as follows: "The density of the right side of the sphenoidal sinuses indicates infection. The optic foramina are normal in size. There is no evidence of increased intracranial pressure. The superior strait of the sella turcica is closed by fusion between the

clinoidal processes. This is of relatively frequent occurence."

On the following day the right field showed a relative scotoma in the formerly clear central area. A portion of the right middle turbinate was removed and the right posterior ethmoid and right sphenoidal sinuses were opened, with discharge of a slight amount of pus. A noted erable enlargement of the blind spot (Bjerrum and campimeter). The patient was examined by Hofrat Wagner-Jauregg, who found slightly diminished reflexes, abdominal sluggish. X-ray studies were made by Professor Schüller, who found the right sphenoid still cloudy; the sinus appeared to extend into the pterygoid process.

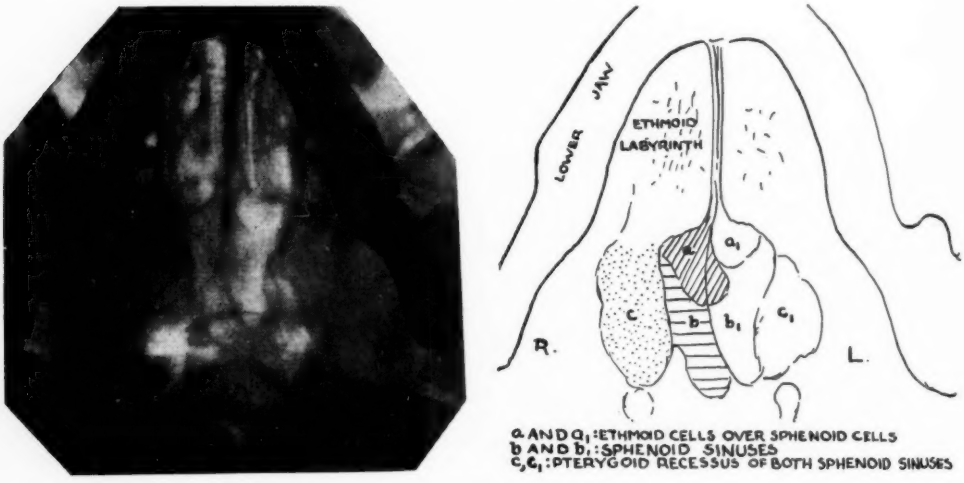

Fig. 1 (Elles). X-ray study of sphenoid region with explanatory diagram by Dr. Schüller.

on the 31st of July indicated that since the sinuses were opened, the spot before the right eye had gradually become much smaller until it was only a small yellowish spot. P.P. 30 cm. Vision (with his correction) in the right eye was 20/30.

On August 9, 1933, there was still some segment remaining of ring scotoma in the vertical and horizontal meridians. Two days later, except for some enlargement of the blind spot, these had disappeared, but paralysis of accommodation still remained. Soon after this the patient left for Europe and joined me for consultation in Vienna.

Prof. A. Fuchs examined the patient on September 11th, and found an accommodation paralysis of both eyes, P.P. 30 cm.; in the right eye a central scotoma for the smallest red target and a consid-

On the following day Professor Oskar Hirsch examined the patient, and it was decided to open the sphenoid further. By submucous route, as the patient had a slight right deviation of the septum which was removed, the sphenoid was entered. A complete horizontal partition was found, below which was a large cell and cells extending into the pterygoid process. Some yellowish fluid discharged. Both sphenoidal sinuses were opened and the cavities thrown into one.

Within an hour the patient was given a book and could read small print at the ordinary near point, even without his distance correction.

Two days after the operation, the vision without glasses was 20/15 O.U., the accommodation in both eyes was equal; the P.P., 11 cm.; the fields of

vision normal; the right blind spot was still enlarged for the smallest red target.

On June 14, 1938, the vision without correction, was O.U. 20/20; with correction, O.U., 20/15. The fundi were normal; the fields normal; the P.P. 11 cm.

Discussion

The rapid clearing of both lesions is an astonishing thing. One might expect the scotoma due to the retrobulbar neuritis to clear rapidly, but scarcely the accommodation paralysis.

De Schweinitz[1] reported homonymous crescentic scotomas in association with ethmoiditis but with no abnormality of accommodation. There was rapid reduction in the size of scotomas. He called attention to the close relation of the optic nerve and the posterior ethmoids and sphenoid, believing the edema with pressure on the nerve to be the *modus operandi* in cases of sinus infection with scotoma, and that the crescentic lesion may be the remains of a ring scotoma, since the latter are not infrequently associated with ethmoid and sphenoid disease.

Franchere,[2] in an observation on the cause of accommodative weakness, reported cases due to nasal obstruction with immediate return of accommodation when the nose was shrunk with cocaine.

Duane,[3] in considering anomalies of accommodation clinically, mentioned toxic conditions and nasal obstruction. In discussion, Zeigler said that the most common cause was pressure of a turbinate against the septum.

Van der Hoeve,[4] in writing of the optic nerve and accessory sinuses, described a central scotoma and enlargement of the blind spot. He stated that annular scotomata are less commonly found, and that the field changes clear up under treatment. He reported a case of retrobulbar neuritis not cleared up by sinus operation but in which, by X-ray examination, a residual infected ethmoid cell was found; drainage relieved the condition.

Traquair,[5] in the Doyne lecture on "Scotomata and their interpretation," said that, broadly speaking, causes of scotomata are nutritional: (1) Toxic and inflammatory. There is rapid onset and usually a high degree of recovery, suggestive of nerve-fiber or ganglion-cell lesion. There are no hemianopic features. (2) Pressure. This form is insidiously progressive. Usually there are peripheral field changes, also. Often hemianopic, the external pressure may interfere with the blood supply.

Suker,[6] in considering paralysis and paresis of the muscles of accommodation, called attention to the division of the third-nerve nucleus, the most anterior part, the accommodation center. The next, the center for iris contraction; and the posterior, the center for the extraocular muscles. The blood supply for the two former centers is separate from that of the extraocular-muscle center. He called attention to irritation due to nasal pathology.

C. A. Veasy[7] reported five cases of paresis of accommodation, due to focal infection, which cleared up rapidly by removal of the foci.

R. Lindsay Rea[8] quotes van der Hoeve in describing a typical form of scotoma showing annular scotoma due to accessory-sinus disease. He says that Professor de Grosz of Budapest found, in 18,587 patients, 58 cases of optic-nerve diseases of nasal origin.

Greenfield Sluder[9] described a specimen in which, in the sphenoidal fissure, the oculomotor, abducens, and ophthalmic nerves were exposed to an eggshell separation from the sphenoidal cell. The third and fourth nerves are in close association with the anterior clinoid processes or lesser wing of the sphenoid

which are not infrequently hollowed out by a prolongation of the sphenoid sinus or (from in front) by an extension from a posterior ethmoid cell bringing these nerves into close association with these cells.

He reported a case of complete paralysis of accommodation of the left eye in a man, aged 35 years, of six months standing. There was present a sphenoiditis of low grade with moderate hyperplasia and very scant mucopurulent or mucous secretion. Accommodation began to return one month after operation and rapidly recovered. There was no dilatation of the pupil.

Sluder[10] again stated that optic-nerve disease secondary to sinus disease is too well known and proved to require arguments. He could see no reason why we should not assume as much for the other nerve trunks in close association with these sinuses.

Peters,[11] in his consideration of the ocular symptoms in the diagnosis of obscure chronic sinus disease, suggested that the reason the scotomata due to papillomacular-bundle involvement are more frequently observed is that, although this bundle lies centrally, the nerve fibers are more highly specialized and delicate in structure. He also reported unilateral loss of accommodation.

Brawley[12] called attention to a class of cases in which no pus could be detected in the nose at any time, except in a few cases which showed muco-pus subsequent to probing and irrigating. Pressure of the congested tissue on the contained nerve endings, together with stasis, produced symptoms and resulted in reflex vasomotor disturbances in the circulation of neighboring structures. Insufficiency of accommodation and convergence is usually observed, and there may be a reduction of vision.

According to Onodi,[13] in dealing with accessory-sinus suppuration as a cause of visual disturbance, we have to consider the nature and spread of the infection, and the complications due to infection of the blood vessels, as well as the presence of any bone dehiscence. He[14] stated that, although the majority of visual disturbances caused by accessory-sinus disease have been unilateral, a double affection arising from disease of the posterior ethmoidal cells or sphenoidal sinus of one side is possible, as can be proved by morphological observations, as he has indicated.

Accessory-sinus suppuration,[14] and pressure on the nerve or on the blood vessels of that part which receives recurrent twigs from the central artery of the retina and which the central vein of the retina joins, may cause a similar condition of central scotoma. Swelling within that part of the optic canal may cause venous congestion and so injure the papillomacular bundle. A venous congestion within the accessory sinuses and nasal cavity may likewise indirectly affect the optic nerve.

That inflammation[15] in the sinuses, by direct extension to the nerve sheath or through the veins, may cause a disturbance of the axial bundle of the optic nerve is easily comprehensible. Toxic effects, too, may produce the central scotoma. It is through its vulnerability to toxins that the lesions of the papillomacular bundle are best known. This explains tobacco amblyopia.

Hoffman,[16] Lapersonne, and Stanculeanu observed oculo-motor paralysis in cases of sphenoidal-sinus suppuration. Moreau and Schröder have described accommodative asthenopia in cases of suppuration of the sphenoidal sinus.

The[17] nasal cavities may appear normal or but slightly altered in appearance, while suppuration is nevertheless present in one or more sinuses, so that only by

careful investigation can a diagnosis be made. In the presence of visual disturbance we are justified in undertaking operative exploration of the posterior accessory sinuses even though they produce no visible intranasal suppuration.

Although cures of optic neuritis and oculomotor paresis have been recorded in cases in which only conservative treatment was carried out, yet in most cases of disease of the accessory sinuses associated with visual disturbance a free opening of the affected sinuses is indicated.

It is difficult to explain the combination of optic neuritis and bilateral paralysis of accommodation, as one thinks of the latter as being due to a nuclear involvement. In early multiple sclerosis, an accommodation paralysis is rare, and one cannot explain the bilateral involvement. On the other hand, in considering an encephalitis, one cannot expect a retrobulbar neuritis.

The rhinological etiology which readily explains the rapid clearing of the retrobulbar lesion after operation does not so easily explain the spectacular return of accommodation within an hour's time. Certainly an intoxication of the accommodation center in the aqueduct of Sylvius could not have disappeared so promptly.

An edema of the mucosa in the sphenoid, particularly in view of the anomalous development, with, no doubt, marked thinning of its walls, bringing not only the optic, but the fibers of the third nerve for accommodation into a vulnerable position, might be considered as a causative factor. Or by such relief of pressure a subsidence in a reflex irritation in the nerve would account for rapid recovery.

A functional component is out of the question, in view of the careful examination and accurate information.

Summary

1. This case presents the unusual combination of two ocular symptoms: right retrobulbar neuritis and bilateral accommodation paralysis, with X-ray diagnosis of right sphenoiditis in an anomalous sphenoid.

2. Multiple sclerosis and encephalitis were ruled out as etiological factors.

3. There was absence of rhinological evidence of sinus disease.

4. The importance of free opening and exploration of the sinuses in visual disturbance is stressed.

Niels Esperson Building.

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THE SPEED OF PICTURE RECOGNITION AND THE SPEED OF WORD RECOGNITION IN CASES OF READING DIFFICULTY

THOMAS H. EAMES, M.D.
Boston

Probably the majority of cases of difficulty in reading are educational in nature and respond readily to a well-planned program of remedial reading. Those that do not respond have occupied the attention of educators and physicians for some years. Psychiatrists and psychologists have leaned toward theories of emotional and mental disturbances of various sorts, educators have investigated the weak points in the various methods of instruction and have devised very efficient remedial techniques which are often helpful, oculists have considered visual and ocular defects and diseases as possible inhibitory factors. In this they have been joined by a number of educators, psychologists, and others. In short, no specific cause of poor reading has been found, and at present there is no single remedial approach that is uniformly effective. It is necessary to treat the whole child rather than various parts of him, but it is also necessary to investigate the functional and pathological processes affecting the parts in order to understand the problem in its entirety.

Pursuing this approach the author, his colleagues, and others have been investigating the relation of ocular factors to poor reading for several years and have reported their findings in medical, educational, and other professional journals. Recently interest has centered on the speed of recognition. It seemed probable that poor readers might be retarded in their speed of recognizing letters and words. An initial study[1] was made with a tachistoscope composed of a visual-education projector to which had been fitted a shutter with speeds varying from 1.00 to 0.01 second. The words were photographed on 35-mm. moving-picture film and were projected, one at a time, in measured exposures. The projection screen used was about the size of a textbook page and was placed at the usual reading distance before the child. The letters and words projected on the screen were slightly larger than ordinary primer type.

Ninety subjects were included in the initial study, of which 50 were children having trouble in reading and 40 were children without reading difficulty. It was found that the group of poor readers exhibited a slower median speed of word recognition than did the other group.

The maximum speed of the shutter, although adequate for study of the group of poor readers, was found to be too slow for most of the members of the group without reading trouble, and the need for a faster and more accurate shutter was exhibited. Accordingly, a shutter with a speed range from 1 to 0.0033 second was secured and was rebuilt by a shutter expert for greater precision and accuracy.

The possibility that poor readers might recognize words at a slower speed than they would recognize objects presented itself and was investigated in the study to be reported in this paper. The recognition of words involves acts somewhat different from those involved in the recognition of objects or pictures. In recognizing a picture or object one fixates any part of it and then explores in any direction, from right to left, from left to right, up, down, or in any way he wishes. The components may be examined in any order, direction, or combination of directions that may chance, and efficient recognition of the object may be secured. The

recognition of an object involves only visual perception with association of the name of the object. Other associations may occur but they are not included in the process *per se*.

Recognition of words is more complex. It involves inspection of the word in a definite direction, from left to right, and includes visual perception of the word as an object, association of its name, and association of its meaning.

In the present study a new film was prepared on which the words were arranged, one to a frame, as before. The words were selected from the Gates, Thorndike, Gates-Huber, Elson-Gray, and Boston University word lists. All words selected occurred in approximately the same position on those word lists arranged in order of appearance of the word in the teaching of reading.

To measure the speed of recognition of objects under comparable conditions it was decided to use projected photographs of common objects such as trains, boats, dogs, cats, fire engines, and so on.

The technique of measurement was as follows: A few pictures or words were flashed to show the child what to expect. Next, words were flashed, beginning at the highest speed of the shutter and working downward to a point where the words were recognized consistently. This speed was taken as the speed of word recognition. Only words which the child knew, as indicated by subsequent reading of the list, were counted in the determination of the speed of word recognition. To use words which were unknown to the children would have been to measure the speed of word analysis rather than the speed of word recognition.

The speed of picture recognition was determined by starting at the fastest speed and flashing pictures on the same screen, gradually reducing the speed of the shutter until a point was reached at which pictures were recognized consistently. This was taken as the speed of picture recognition.

In determining both the speed of word recognition and the speed of picture recognition each word or picture was flashed but once to avoid practice effect.

The speed of picture recognition and the speed of word recognition were measured in the cases of 150 school children, of whom 100 were having trouble in reading, and 50 were without reading difficulty. Thirty of the children in the latter group had eye trouble but no educational trouble, and 20 were without either eye or educational difficulties. All cases were given a complete eye examination.

TABLE 1

CENTRAL TENDENCY

	100 Poor Readers	50 Subjects Without Reading Trouble
A. Speed of Picture Recognition.		
Median	0.0141 sec.	0.0033 sec. or faster.
Q_1	0.0667 sec.	0.0148 sec.
Q_3	0.0033 sec. or faster.	0.0033 sec. or faster.
B. Speed of Word Recognition.		
Median	0.0295 sec.	0.0033 sec. or faster.
Q_1	0.3200 sec.	0.0125 sec.
Q_3	0.0105 sec.	0.0033 sec. or faster.

A study of the speeds showed skewing of the curve in three of the four main distributions. This occurred at the faster end of the range and indicated that the new shutter with its faster speed was not rapid enough to measure all of the cases, some having speeds probably in excess of 0.0033 second. For this reason it was necessary to employ the median as a measure of central tendency because the use of the average, as is common in medical literature, would produce unrepresentative results, the exact speed of the cases falling beyond 0.0033 second being unknown. The median is not influenced by such cases when it falls elsewhere

than in the initial or final speed interval. The median represents the mid-point of the distribution and is used more often than the average in educational investigations. Q_1 and Q_3 referred to in this paper relate to the quarter points of the distribution.

As in the earlier study it was found that the poor readers exhibited a slower median speed of word recognition than those without reading trouble. In addition there was shown a tendency on the part of the poor readers to have slower median speed of picture recognition as well as a slower speed of word recognition. The distributions for the group of poor readers were far enough toward the slower end of the speed range so that both medians and all but one of the Q's were calculable, but the group of cases without reading trouble, being farther up the speed scale, fell within such a range that both the median for the speed of picture recognition and the median for the speed of word recognition fell beyond the fastest speed of the shutter and only Q_1 of each distribution could be calculated with accuracy. In the group without educational or eye trouble only a few cases fell below the upper speed limit of the shutter and the median and Q's all fell at or above 0.0033 second.

TABLE 2

FREQUENCY OF TYPES

	100 Poor Readers	50 Subjects Without Reading Trouble
	percent	percent
Type 1	7	50
Type 2	18	10
Type 3	49	24
Type 4	1	12
Type 5	25	4
	100	100

The facts brought out by the relative positions of the distributions along the speed scale include the tendency for the poor readers as a group to be slower in both picture and word recognition than the children without reading trouble, together with the tendency for the children with eye trouble but without educational difficulties to be slower in both picture and word recognition than the children with neither eye nor educational trouble. There is an implication that eye trouble may act to retard both the speed of picture recognition and the speed of word recognition and therefore the speed of recognition in general.

Certain types of cases were noted in comparing the speeds of picture and word recognition in both groups. They were classified and assigned mathematical limits based on the findings of the earlier study and on clinical observations. Cases with measured values falling outside the limits of the classification were grouped as unclassified. The classification and its limits were as follows:

1. Speed of picture recognition and speed of word recognition both 0.0033 second or faster.
2. Speed of picture recognition 0.0033 second or faster. Speed of word recognition slower than 0.0100 second.
3. Speed of picture recognition and speed of word recognition both slower than 0.0100 second.
4. Speed of picture recognition slower than 0.0100 second. Speed of word recognition 0.0033 second or faster.
5. Unclassified.

Type 1, in which the speeds of picture recognition and of word recognition were both 0.0033 second or faster, was about seven times as frequent among the children *without* reading trouble. The frequencies of types 2 and 3 among poor readers were about twice as great as among children without reading difficulties. Type 3, in which the speed of picture recognition and the speed of word recog-

nition were both retarded, was predominant in both groups.

It appeared worth while to investigate the frequencies of eye trouble in the various types of cases and this was done. Refractive errors were counted only when of .75 diopter or more, and muscular imbalances were included only when of 6 or more prism diopters.

The frequency of eye trouble was not very different in the two groups but the poor readers presented the greater incidence. They also exhibited greater frequencies of hypermetropia, muscular imbalance at the reading distance, and deficient fusion for textbook type. This is consistent with the findings of other investigators[2, 3, 4] and myself.[5, 6]

The poor readers falling under type 2, in which word recognition was retarded and picture recognition was not (according to the limits of the classification), presented a higher incidence of deficiencies of fusion for textbook type.

In type 3, wherein both the speed of picture recognition and the speed of word recognition were retarded, the poor readers showed greater frequencies of hyper-

TABLE 3

FREQUENCY OF EYE TROUBLE

100 Poor Readers	50 Subjects Without Reading Trouble
66 percent	62 percent

TABLE 4

EYE CONDITIONS EXHIBITING A DIFFERENCE IN FREQUENCY BETWEEN THE GROUPS OF 5 PERCENT OR MORE

100 Poor Readers		50 Subjects Without Reading Trouble	
A. The Entire Groups			
Hypermetropia	14% more*	All kinds of eye trouble differing within the above limits occurred less frequently in this group	
Muscular imbalance at the reading distance	5% more		
Deficient fusion for testbook type	8% more		
B. According to Types			
Type 1. 7 Cases		25 Cases	
Amblyopia	6% more	Hypermetropia	16% more
Hypermetropic astigmatism	6% more	Myopia	8% more
		Myopic astigm.	8% more
		Exophoria	6% more
		Convergent strabismus	20% more
Type 2. 18 Cases		5 Cases	
Deficient fusion for textbook type	11% more	Hypermetropia	18% more
		Amblyopia	20% more
		Convergent strabismus	15% more
Type 3. 49 Cases		12 Cases	
Hypermetropia	42% more	Myopia	13% more
Muscular imbalance at the reading distance	10% more	Amblyopia	9% more
Type 4. 1 Case		6 Cases	
Restricted fields	100% more	Hypermetropia	33% more
		Amblyopia	17% more
Type 5. 25 Cases		2 Cases	
Exophoria at the reading distance	44% more	Hypermetropia	8% more
Deficient fusion for textbook type	12% more	Hypermetropic astigmatism	29% more
		Myopia	8% more
		Myopic astigm.	12% more

"% more" indicates the amount by which the condition is more frequent in the group than is the frequency of the same condition in the other group.

metropia and of muscular imbalance at the reading distance.

Only one poor reader fell under type 4. This case exhibited restricted fields but nothing more. A number of cases without reading trouble occurred in this type and presented various eye conditions.

Deductions based on clinical experience indicated that probably type-2 cases were handicapped primarily by educational factors, the speed of picture recognition being at the upper level and the speed of word recognition exhibiting retardation. This would suggest an associative deficiency that might be physical but would be more likely to originate in the educational experiences of the child. The lower frequency of most forms of eye trouble among the poor readers is consistent with the view that the eyes are probably not primarily involved in most cases of this type.

Type-3 cases were thought to be the ones most likely to be influenced by eye trouble, especially as *both* the speed of word recognition and the speed of picture recognition were retarded. Some evidence in support of this hypothesis is presented by the greater frequency of eye trouble appearing in this type in both groups. The fact that the poor readers displayed a frequency twice as great as the other group suggests that retarded speed of recognition is an important factor in poor reading. It also emphasizes the need of a complete eye examination in all such cases.

It was regarded as improbable that type-4 cases, wherein only the speed of picture recognition is retarded, would be influenced by eye trouble and the condition was attributed to too much short-exposure drill with flash cards, tachistoscopes, and other devices intended to increase the speed of reading, which were believed to have increased the speed of word recognition above an initially low general-recognition speed. Only one of the poor readers fell under this classification type but a few of the children without reading trouble came under this heading and presented a considerable incidence of eye troubles. Although this gives no true indication of the cause of this type of case, partly because of the small number of cases and partly because we have but one poor reader for comparison, we may infer that type-4 cases are infrequent among poor readers and probably bear little or no relation to reading difficulties. The educational explanation of the type seems reasonable and merits consideration. Speeds of word recognition in excess of the speed of picture recognition can be secured experimentally by short-exposure drill. In such cases the tendency is toward mechanical word calling rather than true word recognition.

Cases of type 1, in which both the speed of picture recognition and the speed of word recognition are at the upper speed limit of the shutter are infrequent among poor readers as would be expected. The fact that some of the poor readers fell in this class emphasizes the danger of attributing poor reading to any single cause. Clinical experience indicates that poor readers exhibiting characteristics of type 1 are likely to make numerous errors and may be retarded in various aspects of reading accomplishment, but they are less likely to display retardation in the *speed* of reading than are subjects belonging to other types. That half of the cases of those without reading trouble were of type 1 is surprising only in the lowness of the incidence, a somewhat higher percentage having been expected.

The unclassified cases, type 5, included various individuals exhibiting tendencies similar to those in other types but with speed values such as to exclude them. One would expect to find a fair representation of the eye conditions disclosed in the other types and this is what the data show.

Treatment of the poor reader who exhibits retarded speed of recognition should include correction or treatment of all existing eye defects and diseases. In addition the teacher may be advised to give short-exposure drill, especially in the cases of types 2 and 3. Cases presenting retarded speed of word recognition without retardation of the speed of picture recognition, type 2, are likely to respond best to short-exposure drill. This is carried out easily by the teacher by means of flash cards which she can make herself or secure from any school supply house. She may also use a simple paper or pasteboard tachistoscope which she can make or purchase for a small sum. By these devices, words that the child knows are flashed for recognition at shorter and shorter exposures. Later phrases are presented in the same way and finally the phrases of a complete sentence are flashed successively.

Recently, Dr. W. F. Dearborn and Dr. I. H. Anderson, of the Harvard Graduate School of Education, exhibited some excellent 16-mm. films employing the short-exposure principle. Dr. D. D. Durrell, of the Boston University School of Education, has developed a simple tachistoscope which is available at very low cost.[7] I have used the measuring tachistoscope with which the data for this study were collected as a short-exposure-drill apparatus and have found it satisfactory. Projection film strips are made readily by photographing the words and phrases with an ordinary miniature camera. Other devices intended for short-exposure drill and possessing various degrees of ultility continue to appear. My preference is for the types mentioned in this paragraph.

Summary

Eye examinations and measurements of the speed of picture recognition and the speed of word recognition were made in 100 cases of school children who were having trouble with reading, and 50 cases of school children who were without reading trouble. It was found that the poor readers tended to exhibit slower speeds of both picture and word recognition and therefore of the speed of recognition in general. The children without reading trouble but with eye difficulties tended to be slower in the speed of recognition than did a group of 20 school children who were without eye or reading trouble. Some support was found for the hypothesis that eye defects may retard the speed of recognition. The eye conditions more common among readers were found to be hypermetropia, muscular imbalance at the reading distance, and deficient fusion for textbook type.

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THE PROBLEM OF STATISTICS RELATING TO BLINDNESS AND THE BLIND*

HARRY BEST, PH.D.
Lexington, Kentucky

In practically all scientific inquiries at the present day, especially those that to a greater or less extent are of quantitative character, there is imperative need of statistical data, both to present something of a picture of conditions as they are, and to serve as some sort of a measure of progress from one period of time to another. Possibly in no field of public or social welfare, or in what we call the social sciences, is the importance of statistics more pronounced than in the matter of blindness and the blind—in the matter of those suffering under what is regarded as the heaviest of human afflictions. To deal adequately with the problem, including that of the prevention of blindness, statistics are loudly called for. We need them in order to know the number of the blind in our midst, and different characteristics regarding them, as their age, the age at the occurrence of their blindness, their numbers in respect to sex and to race and nativity, their marital condition, their economic condition, the extent of education among them, their ability to read raised print—and, above all, the causes of their blindness. We can hardly be expected to know how to deal with the problem of blindness or how to make advance in our treatment of it unless we have this essential information in our hands.

The principal statistics in the United States, and the only ones of nation-wide bearing, relating to the blind are those of the United States Census Bureau, which have been collected for 100 years, the first returns being for the year 1830. As the decades have passed, these returns have in general taken on a wider range, with inquiry as to various matters besides bare enumeration. Other statistical data which we possess have been of a more restricted nature, limited to local geographical areas. Such statistics are those of schools for the blind, agencies for the adult blind, medical periodicals, industrial-accident boards, and health authorities—together with the findings of some special nation-wide organization concerned with blindness.

Statistics from schools, especially so far as they relate to the causes of blindness, are far from satisfactory. They have not always been compiled according to scientific principles, and there has often been wanting reliable ophthalmic examination. (In not a few cases the parents have themselves been ignorant, and have been unable to give the exact cause of blindness.) Of later years better and more precise methods of classification have been employed in a number of schools. Certain agencies for the blind have also conducted investigations, some being better than others; not infrequently there are considerable variations in the figures presented. There have been some very good investigations on the part of ophthalmologists, these always being superior and more reliable in the matter of the causation of blindness, the chief defect with some of these being in the smallness or the limitation of the number of cases involved. Statistics as to accidents collected by industrial-accident boards and similar organizations would appear, within their restricted range, to be fairly trustworthy. Statistics with regard to causes of

* From the Department of Sociology, University of Kentucky.

blindness collected and printed by some such body as the National Society for the Prevention of Blindness are, with its great care and high scientific procedure, always of the utmost value.

Differences in the returns between local investigations and census enumerations are doubtless in part due to longer periods of time for the former, to wider acquaintance and a fuller coöperation on the part of neighbors, and to less fixed or less standardized definitions of blindness (often including more liberal conceptions). The most satisfactory presentations are likely to be found where the most intensive work for the blind is carried on.

Criticism has sometimes been directed towards the census statistics. It is asserted that the figures presented are both incomplete and inaccurate. This charge is doubtless true to a greater or less extent. The most practical consideration here, however, is whether these statistics are of such character that they may be used, with due explanations and interpretations, to form some sort of picture of the situation respecting blindness and the blind in the United States that can be of service to us. Our immediate inquiry should be whether they have enough completeness and accuracy, relatively speaking, to make them of a certain genuine usefulness to us as we try to understand fundamental factors in what is involved in the great affliction of blindness.

Without doubt the number of the blind reported in the Federal Census is understated. Nearly all local investigations disclose larger numbers in their respective areas. From them we reach the conclusion that there are hardly less than 100,000 blind persons in the United States, a number considerably in excess of that given in the Federal figures. This, however, is probably the chief error, or the least misleading feature, of the Federal returns; its seriousness is much reduced when checked by the returns of local inquiries. Even the broad enumerations of the blind at the several census years, while possibly to some extent upon different bases, do with little doubt show general trends.

It is possible that the census returns for the blind may not be equally incomplete over all the country; but we have no evidence that these statistics are grossly unequal, or so much so as to deprive them of value. Even though the census figures may not be altogether representative with respect to such matters as age, race, occupation, causes of blindness, and so forth, they are very likely sufficiently so to help us get a fair portraiture of conditions respecting the blind in this country. (In the latter matter the census statistics are sometimes based in general upon the returns of special schedules which were sent to blind persons whose names had been included in the original enumeration.)

Let us examine some of the census findings and see how they compare with the findings from other sources—in particular in relation to the causes of blindness—which have presented figures near the time of the census report for 1920. (For the census of 1930 there was secured only the enumeration of the blind, with sex and age.) These sources are: reports of the Minnesota Board of Control, of the Connecticut Board of Education of the Blind, of the California Department of Public Welfare, and of the New York State Commission for the Blind; the Journal of the Missouri State Medical Association; the American Journal of Ophthalmology (for Colorado and Illinois); and reports of the Division of the Blind of the Massachusetts Department of Education (on a somewhat different basis, and relating to new cases referred by the Massachusetts Eye and Ear In-

firmary)—all offering figures quite divergent among themselves. Among the leading causes of blindness are generally recognized accidents, cataract, atrophy of the optic nerve, and glaucoma. In the case of accidents the census figures are higher than most of the other figures; though when we consider accidents directly affecting the eye, the former is found to be not far from the average or the median. With cataract the census figure is surprisingly close to the other figures except as to extremes one way or the other; in the census analysis it is pointed out that the percentage for cataract is not quite large enough, some cataract being erroneously ascribed elsewhere. The census figures for glaucoma are lower than is the case in most of the other investigations; attention is again called to the appreciable amount placed elsewhere. The same observation applies to atrophy of the optic nerve. Ophthalmia neonatorum in the census has a middle position among the other investigations. The remaining causes do not permit comparisons, some in consequence of the smallness of the figures. At any rate, it appears, the census figures are found not to be farfetched or obviously wrong.

If we compare the percentages of blindness due to the several causes in the census years 1910 and 1920, we find a very decided similarity among those that are comparable—some of the changes being chargeable to actual decline in the proportions for the two periods. This is true also, though to a less extent, when comparisons are made with the year 1890.

If we now turn to the proportions of the blind according to age at different census years, from as far back as 1860, we discover few great changes, percentage declines being most in evidence in the early years of life, just as we would expect in view of the more pronounced conquests over blindness at that time of life. The same general comment may be made with respect to the proportions of the blind according to age at occurrence of blindness for the years given; namely, 1880, 1910, and 1920. If we pass on to other matters affording comparison as between the census years 1910 and 1920, as sex, race and nativity, marital condition, economic condition, and ability to read raised print, we still find notable likenesses, the changes that take place being only what we would naturally expect. The one exception has to do with the matter of education, in respect to which there appears to have been considerable misunderstanding on the part of those replying to questionnaires. Due account of all this is taken in the census interpretations.

It might be added that the findings obtained by the census investigations are, with the exception of the matter of the total number of the blind in the country, not greatly at variance with the findings of other investigations for more restricted areas and are to a considerable extent borne out by them. It might be stated, too, that the census findings are largely in keeping with what those who have been concerned in work for the blind would expect (except once more as to total numbers). Even with regard to such an intractable matter as the causation of blindness, the census figures do afford us rough approximations, or at any rate a clue to an understanding of the situation. If the census findings have done these things, they are entitled to some measure of our regard—and with respect to a matter of most baffling character, and one of which we were about to despair.

It is not contended that the statistics of the Census Bureau on the blind are complete or accurate in all respects; far from it. But it does not seem unreasonable to believe that, even though approximations, they are not far wide of the mark,

or are seriously misleading. It can hardly be questioned that they do afford some guidance to the actual situation regarding the blind; they do throw material light on their condition. They are certainly sufficient to provide us with working material in our efforts to understand their problems and their needs—certainly sufficient to help us do our duty by them and to render them genuine service.

It might also be said that it may be many years before we have so rich a collection of statistical matter relating to the blind, even though later years might show greater accuracy or completeness, as we have had in the census reports for 1920 (and also for 1910). Obtaining satisfactory statistics regarding the blind will never be an easy matter; so far as a nation-wide investigation is concerned, the obstacles in the way will remain formidable—possibly we can hardly foresee a time when we can expect to get statistics on a far-extended scale that will meet all our rigid requirements.

A note of high encouragement on the present state of our inquiries regarding the blind lies in the fact that there is now in the field a special organization, one that we should have had long ago—the Committee on Statistics of the Blind—which has a fertile if pioneer field before it. Its creation is to be regarded as one of the most constructive and far-sighted measures ever adopted in the work for the blind or in the prevention of blindness in the United States. It is drawing up special report forms and charts, not necessarily at present requiring detailed ophthalmological record, but the minimum of essential medical data. It is stressing underlying factors in the causation of blindness as well as the seat of the eye troubles. Thus both an etiological and a topographical classification of the causes of blindness will be provided all according to standard classified medical nomenclature.

We must keep in mind that statistical knowledge regarding the blind, and especially regarding the causation of blindness, would be greatly furthered, in fact immensely advanced, if a registration could be kept of all persons becoming blind within each year, with the cause assigned for each case, and with the individual characteristics afforded in each case.

There must be insistence upon the value to us, the indispensability, of statistics regarding the blind—statistics being, as they have been called, instruments of precision in dealing with our problems, social and other. Till we can find better ones, we shall have to make intelligent use of those now in our hands, which are not only the best that we have, but all that we have. What is far more important, we must know that there is a heavy duty resting upon us to have full, accurate statistics relating to the blind, as full and as accurate as it is within human powers to secure; with them all there must be the utmost of ophthalmological advice. Perhaps the very defects and shortcoming and imperfections of the statistics we have considered will spur us on to so worthy and so noble an objective.

NOTES, CASES, INSTRUMENTS

GONIOSCOPY AS AN AID TO LOCALIZING SMALL FOREIGN BODIES IN THE ANTERIOR CHAMBER

A. W. McALESTER, III, M.D.

Kansas City, Missouri

The gonioscope lens has proved a very valuable aid in localizing small foreign bodies in the anterior chamber. It should be used routinely on any penetrating wounds of the anterior segment of the globe, regardless of whether the puncture has been a clean one, not carrying foreign material into the eye, or there has been a laceration from a piece of slate; that is, where there has been ample opportunity for small foreign bodies to be deposited in the anterior chamber and thence reach the filtration angle.

This means of examination has been found most valuable in localizing glass in the anterior chamber. The writer has recently had the opportunity of examining five eyes that were lacerated by the breaking of spectacles, or the shattering of windshield glass. Two of the eyes had been irritated for over a period of three months prior to the date of our first examination. Slitlamps and oblique examination revealed no foreign material in the anterior segment of the globe. However, in each instance when a gonioscope lens was used, small spicules of glass were seen lying in the filtration angle in the 5:00 to 7:00-o'clock position. This procedure in connection with the examination greatly aided in the removal of these foreign bodies.

It has been our observation that multiple small foreign bodies, less than one mm. in any direction, tend to gravitate into the filtration angle. Their progress can be determined by repeated gonioscopic examinations over a period of several months. Such foreign bodies as I referred to were carbon or slate.

One case has been observed in which a foreign body apparently became encapsulated in the filtration angle. The history was that of a wire puncture 15 years prior to our examination. The eye was quiet, having good vision, and there was no indication for interference.

We find that the ordinary three power Zeiss loupe has enough magnification and the Hammer light sufficient illumination for such examinations. The microscope attachment of the slitlamp is not necessary for this type of work.

2003 Bryant Building.

SOCIETY PROCEEDINGS

Edited by Dr. H. Rommel Hildreth

SAINT LOUIS OPHTHALMIC SOCIETY

January 28, 1938

Dr. Lawrence T. Post, *president*

Studies on inclusion blennorrhea

Dr. Adolph Lange (as co-author with Dr. L. A. Julianelle) read a paper on this subject which was published in this Journal (August, 1938).

Discussion. Dr. L. Julianelle said that when they first started this study, they were primarily interested in working out any possible relationships between trachoma and inclusion blennorrhea. They found such unsatisfactory information in textbooks and in the literature in general on the clinical aspects of the disease that they decided to undertake a clinical study as well. While inclusion blennorrhea is an interesting disease, it is relatively rare, so that they saw only 22 cases over a period of five years. They have been interested in the etiology, and while it is not desirable to make a report at the present time, the indications are that the causative agent is a filterable virus. Thygeson states that the virus is the elementary body associated with the epithelial-cell inclusion. They have no evidence on that point. Dr. Harrison, Dr. Lange, and he have no evidence to prove that the elementary body and the virus in inclusion blennorrhea are the same. In the laboratory they made certain collodion filters of such pore size (0.6 p.) as to retain bacteria but still allow the virus to pass. They could not be certain that such filtrates contained elementary bodies even though they were infectious for monkeys.

Answering Dr. Jacobs, he said that the original Giemsa stain calls for an overnight staining period. The Wright's stain has all the advantages of the Giemsa stain in showing the inclusion bodies, and in addition a preparation is ready for examination in only 10 minutes.

In answer to Dr. Gross, he stated that in inclusion blennorrhea the inclusion body is found in a scrape smear, but is not found in folliculosis. Inclusion blennorrhea can be differentiated further because of its infectivity for monkeys, while folliculosis is noninfectious.

Dr. B. Y. Alvis said he remembered hearing Dr. Lindner discuss this disease when he was here several years ago. He emphasized the similarity of the inclusion bodies found in the infant's conjunctiva, the vaginal epithelium, and in trachomatous conjunctivae. Dr. Julianelle had mentioned the fact that in many communities the disease is not well known among the oculists. Dr. Alvis believes that none of those who attended Lindner's course here would fail to think of inclusion blennorrhea when seeing an infant showing a mild persistent mucopurulent blennorrhea. This seems to him a good illustration of the value of having leading teachers from other centers come to give courses of instruction.

Nonoperative treatment of cataract

Dr. G. H. Poos read a paper on this subject.

Discussion. Dr. W. H. Luedde said that he was not willing to join a group, no matter how numerous, which is satisfied to sit back in uninformed or unsuspecting credulity and say to every patient with incipient cataract, "Go home and wait until you are ready for operation."

It was just such a statement by one of the founders of this society which brought an old lady, aged 78 years, under his care

in July, 1907. Her distance vision was improved to 15/24 O.D. by a −2.0 D.sph. and to 15/48 O.S. by a −4.0 D.sph. With an addition of +3.75 D.sph. she was enabled to read and sew comfortably.

Ophthalmoscopic examination confirmed the diagnosis of cataract. Instead of submitting to operation for cataract that fall, as she had been advised, this patient returned nine months later, April, 1908. She stated that she had been ill during the winter and that her vision was not so good but that she could still read and sew. Tests with her glasses showed for the right eye vision was reduced to 16/48+ and for the left eye 16/75. The right eye could be improved to 16/30 by adding —1.0 D. sph. to the —2.0 D. sph. of the present glass for the right eye. The vision of the left eye was not improved by addition. Ophthalmoscopic examination indicated a marked increase in the cataract in both eyes. For treatment of a mild chronic conjunctivitis, a solution of zinc sozoiodolate 0.2 percent was prescribed for instillations once daily. Within a month vision with her own glasses was improved to 16/24 for the right eye and 16/48 for the left eye. Three years later (September, 1910) the instillations of the 0.2-percent solution of zinc sozoiodolate were increased to twice daily and in August, 1911, the strength of the solution was increased to 0.4 percent. On November 21, 1913, more than six years after the time originally fixed for her cataract operation, her vision with —5.0 D. sph. was 16/30 right eye, and 16/75+ left eye. She now preferred to read without glasses.

In 1916, after a cerebral hemorrhage and general debility had rendered her practically bedfast at the age of 87 years, she was still able to read J 1.5 slowly at five or six inches, when examined at her residence, and she was receiving daily instillations of the zinc sozoiodolate solution. She died shortly after that.

One explanation for the apparent benefit obtained from its constant use may be the presence of iodine in this zinc salt which, as the essayist has stated, has been used by several observers—notably Louis Dor and A. von Pflugk.

The arrest of a progressive cataract in this first patient was too striking to be ignored. Instillations of zinc-sozoiodolate solution have given similar good results dozens of times though not in all cases. One patient now in her ninetieth year has been under constant treatment and close observation for 23 years. In her case the lenticular opacities covered more than the lower half of the lens.

The indications are that a variety of methods have been followed by the arrest of incipient cataract, but that no single therapeutic method has a universal specific effect. This might be expected, as there are probably a variety of causes for the precipitation or chemical reaction in the lens protein by which its transparency is impaired. Certain chemicals imbibed or inhaled, autointoxications, toxic substances incident to focal infections, and general diseases may be reasonably suspected as possible etiologic factors in the production of cataract.

Quite a number of cases from his clinical experience might be cited to confirm this statement, because the elimination of these conditions when practicable was followed by the apparent arrest of progressive incipient cataract. He believes the radical elimination of foci of infection, especially those involving teeth, nose, or throat, is as desirable before undertaking a cataract operation as it is to promote the cure of iritis or other intraocular diseases.

The earliest possible elimination of dental foci of infection has apparently repeatedly made a cataract operation unnecessary by the preservation of useful vision without it.

As a case in point, reference may be

made to a farmer from a remote district in the Ozarks. He had an incipient cataract in one eye and a more advanced cataract in the other. After the extraction of several infected teeth, his vision was improved to 20/30+ in his better eye and an operation was not needed. However, he begged to have the operation done as he might not be able to return later when it got worse. Subsequently, with glasses his aphakic eye had better vision than the one with incipient cataract. Several years later an injury to the aphakic eye while chopping wood brought him back to Saint Louis. Inquiry revealed that he never had worn his glasses, but had continued to rely on his unoperated-on eye, in which vision had remained better than 20/30 after the radical elimination of dental infection.

Hence, the recognition of the presence of incipient cataract is to him a challenge for a thorough, comprehensive study of the patient. He prefers even to be suspicious of those apparently nonprogressive sharply defined lenticular opacities which may remain unchanged for many years. They should not be treated but kept under observation at reasonable intervals to be assured of their unchanging passivity. The causes of such opacities may be traumatic or temporary disorders, but recurrences are not impossible. He is unable to accept as valid Shastid's contention that the mechanism of accommodation may damage the transparency of the lens. A comprehensive study of that mechanism should convince anyone that such damage can only rest on unwarranted assumption. He does not see that undercorrection of presbyopia or ametropia of any type possesses any virtue in the arrest or prevention of cataract. Correction of refraction may be more sanely considered in relation to the extraocular-muscle balance and general nervous exhaustion rather than as an etiological factor in the production of cataract. Very slight experience does not qualify him to support the claims of A. E. Davis about lens antigen; nor can he make definite statements about the effect of endocrine disorders or dietary regime, although these possibilities must be considered. More recently, he has tried out the effect of certain vitamins without reaching a final conclusion, although in several instances the intravenous injection of vitamin C (Cebione) solution has apparently been beneficial. This latter therapy has the advantage of apparent harmlessness, which applies also to the iodide therapy, and apparently deserves a trial while we continue to search for better etiological understanding upon which more definite therapy must depend. If we ever can know as much as we ought to know about the causes of cataract, the surgical treatment may become almost an anachronism except as a sequence of trauma. For the present we may say that senile cataracts is no more truly senile than arteriosclerosis, although both are phenomena observed in the aged.

Dr. B. Y. Alvis said in many cases of partial cataract the results of the visual tests vary on repeated examinations. If the initial test happens to give a very poor visual acuity, a subsequent one may show much better vision even though no material change has taken place in the lens. He believes one is justified in prescribing some treatment in certain cases so the patient may not feel hopeless, but rather be encouraged by the fact that something is being done to help him.

He cited the cases of two patients whose condition he had followed for many years. His associates were using dionin when these patients were first examined, on the theory that the dionin stimulates the flow of blood and lymph through the tissues and might do some good by improving the nutrition of the lens. It has

a noticeable subjective effect. It does no harm and may do some good.

One of these patients was a druggist, aged 75 years, with beginning nuclear sclerosis. The patient believed the dionin helped to retard the progress of the cataractous change. His vision changed very slowly and now, at the age of 90, he can no longer leave his home. He can still see dimly and is still using the dionin. Dr. Alvis does not believe the treatment did the cataracts any good, but he believes it gave the old man a great deal of comfort.

The other patient, a man of 60 years, began to have complicated cataract several years ago. His vision varied greatly on successive tests, some times being two or three lines better than others. At the same time the trial lenses giving the best results varied considerably. This patient also has used dionin daily for many years and feels it has helped retard the progress of the cataract. Dr. Alvis believes it has done much to allay apprehension.

On the other hand it has been his teaching that the lens becomes more and more dense and less transparent with advancing years and eventually becomes cataractous even when there is no disease other than old age. Such being the case, he holds that nonsurgical treatment of cataract should be limited to caring for the general health and well being of the patient by removing foci of infection, improving nutrition, caring for constituutional diseases when present, and to such local treatment as may keep the conjunctiva healthy and add to the mental comfort of the individual.

Dr. John Green said he had had a limited experience with subconjunctival injections of 1 :4,000 cyanide of mercury in cortical cataract. In some cases he has observed prompt clearing of interstitial haze, accompanied by equally prompt improvement in vision. This experience led him to believe that lymphatic stimulation can occasionally be helpful.

Dr. L. T. Post said one other reason why we may get better vision one day than another is that the accessory illumination may differ, causing variation in the size of the pupil. If the day is bright, vision may be different from what it is when it is dark outside. There are times also when vision varies, depending on refractile differences through various lens facets. Furthermore, if one is using a special treatment he will be unusually interested and so will urge the patient to exert every effort to see better after initiating the new therapy.

Dr. Max Jacobs said that he was convinced that many cases of incipient cataract improve because the patient's general health is improved. After the patient's pupil is dilated and we see vitreous opacities, we send him to an internist or caution him about his general health. Then when he returns in two or three months and is in better health, we find the eyes are improved.

Essential atrophy of the iris

Dr. Hayward Post read a paper on this subject which will be published in this Journal.

H. Rommel Hildreth,
Editor.

COLLEGE OF PHYSICIANS OF PHILADELPHIA

Section on ophthalmology

January, 1938

Dr. Alexander G. Fewell, *chairman*

A case of parinaud's conjunctivitis

Dr. James S. Shipman and Dr. H. D. Barnshaw presented a boy who had first come under their observation about 19 days previously. The patient gave a history of contact with cats and dogs and a neighbor's goat. At that time he had

follicular enlargement and hypertrophy of the conjunctival tissue. This had been more marked than at present. He had marked adenopathy that appeared to follow the outline of the parotid gland. His studies in the hospital were essentially negative. Cultures were negative. His blood count showed eosinophilia 4. Treatment has been instillation of zinc and washes and X-ray irradiation of the glands in the neck.

They believed this is a case of infectious conjunctivitis, possibly of the Parinaud type. Verhoeff found a leptothrix in 17 out of 18 cases examined. This he believes to be causative. He states that this infection may come from animals, but it is not definitely proved.

The other types of infectious conjunctivitis are: conjunctivitis tularense, caused by the Bacillus tularense; Pascheff's disease, which is found in workers with rodents; and the disease that Boyer and Herrenschwand found—the bacillus pseudotuberculosis rodentium. These diseases are probably the same as conjunctivitis tularense.

This boy should probably have a biopsy of the conjunctiva and an agglutination test for tularemia.

Coloboma of the Macula

Dr. Andrew Knox reported the case of F. L., a girl aged 11 years, who was first examined by him at the Episcopal Hospital on October 27, 1937. Vision O.D. was 20/20; the media and fundi were negative; Vision O.S. was 5/200; the external appearance was normal, the cornea clear; the pupil, round and of normal size, reacted directly and consensually to light and also to convergence. The nerve head showed some pallor, especially to the temporal side; the vessels gave no evidence of pathology. Directly in the macula a circumscribed yellowish-white object was seen, which at first glance appeared to be suggestive of a large exudate, but closer examination revealed a depression. The retina, aside from the depression in the macula, was normal in appearance. Directly surrounding the whitish ball in the macula was a medium-sized ring of pigmented choroid. Two small retinal arteries were seen to cross the area of the coloboma. Slitlamp examination revealed no abnormalities. Congenital defects, such as dermoids or colobomata of the lens or iris, were absent. The influence of heredity in relation to coloboma of the choroid has been emphasized by Dr. C. E. G. Shannon. However, the family history revealed no evidence of any congenital defects of the eyes in other members of the family.

Essential Shrinking of the Conjunctiva (Ocular Pemphigus) with Involvement of All Mucous Membranes

Dr. Joseph V. Klauder presented M. G., a white woman, aged 55 years. He first saw the patient in 1931, and at intervals until 1933, then not again until October, 1937. A lupus erythematosus of the scalp, which first appeared in 1907, is now represented by large, atrophic, bald areas. Starting in 1920 and continuing thereafter, small blisters have appeared and disappeared on the buccal mucosa, on the inner surfaces of the lips, and on the tongue. Mild silver protein (25 percent) was employed in treatment of the mouth lesions for five years. Argyria, which the patient now has, appeared in 1929.

The ocular involvement first appeared in 1929. The patient complained of a burning sensation, a sense of heaviness of the lids, and mucoid discharge. At that time she was treated for trachoma. When Dr. Klauder saw her in 1931 the conjunctivae of the upper lids were smooth and glistening with a velvetlike edema. He regarded the condition as lupus erythema-

tosus, which he now believes was erroneous. These ocular symptoms have disappeared. Now the conjunctivae of both lower lids are dry, the cul-de-sacs are narrowed, the fornices have disappeared. In the right eye there is union of the central part of the palpebral conjunctiva of the lower lid with the bulbar conjunctiva.

At different times he has observed a solitary vesicle on the conjunctiva, also on the buccal mucosa and the inner surfaces of the lip. On the buccal mucosa there are rounded areas of redness. There is union in some places of the buccal mucosa with the mucosa over what remains of the gums. There is considerable recession and shrinking of the gums. The teeth are absent. There are linear and rounded white areas on the buccal mucosa, lips, and tongue resembling lichen planus, also leucoplakia. The nasopharynx and pharynx are normal. There are rounded, reddened areas on the outer surfaces of the nasal mucosa and a similar involvement of the mucosa of the anus. There is considerable shriveling of the vulva. The entrance to the vagina is almost obliterated, the labia minora are united. In the center of this union is a dull red area similar to the mouth lesions, surrounded by a narrow white area. The urethra cannot be identified.

It is unusual that after 17 years there has not been some conjunctival involvement. The disease appears to be expending itself on the mucosa of the mouth, urethra, and vagina.

Dr. Klauder also presented O. E., a white man, aged 57 years, born in Turkey. About 1917 the left eye became mildly inflamed. This disappeared in a short time. Since this time the condition he now presents first appeared. There has been no subsequent attack of inflammation. There have been no skin eruptions, nor history of sore mouth or throat.

The conjunctiva of the lower left lid and the bulbar conjunctiva are dry, can be easily wrinkled. The epithelium over the cornea is dry and opaque. The cul-de-sac is shortened. In one area there is adhesion of the palpebral with the bulbar conjunctiva. The tarsi of both upper lids are shortened. There is no pannus. The vision O.S. is ability to detect hand movements. The palpebral conjunctiva of the right eye is dry. The involvement here is considerably less than in the left eye. The cornea shows no involvement. The vision O.D. is 6/21.

Examination of the skin and mucous membranes, including the meatus, nasal mucosa, and anus, disclosed no pathology.

Tuberculous kerato-uveitis associated with a distinctive type of cutaneous tuberculosis in the Negro

Dr. Joseph V. Klauder said that H. G., a negress, aged 37 years, was first seen in March, 1935, presenting uveitis of both eyes. The blood Wassermann was strongly positive. The vision was O.D. 6/9, O.S. 6/15. The patient was given 50 injections of bismuth, 36 injections of neoarsphenamine, and potassium iodide by mouth, between March, 1935, and November, 1937. In August, 1936, the Wassermann reaction of the blood was negative. In November, 1936, a skin lesion appeared on the right thigh, followed by a gradual development of lesions elsewhere.

On both surfaces of the legs are now seen discoid elevated and infiltrated plaques about 2 cm. in diameter, some with a smooth surface, others atrophic and depressed, still others scaly. There are two plaques on the back, and a few on the lower side of the chest following the lines of cleavage. Five split-pea-sized lesions are present on the forehead, and one on the upper lip. Atrophic remains of lesions are on the scalp.

The patient is well developed but poorly nourished. There is roentgenographic evidence of pulmonary tuberculosis. Slit-lamp examination reveals both corneas to be hazy throughout. There are numerous large and small mutton-fat grayish deposits on the posterior surface of the cornea, and many small grayish proliferations around the pupillary border of the iris. The intradermal tuberculin test 1:1000 was negative. The last recorded vision was O.D. ability to see hand movements; O.S., 6/15. Histologic examination of a skin lesion revealed a typical tuberculous infiltrate with associated giant cells, and necrosis characteristic of bacillary tuberculosis as it affects the viscera.

The cutaneous lesions presented by this patient constitute what appear to be a distinctive type of tuberculosis seen in the American Negro. Dr. Weidman and he first called attention to this (Klauder, J. V., Multiple sarcoid-like granulomas of the skin of undetermined nature. Arch. Dermat. and Syph., 1925, v. 12, Aug., p. 171; Klauder, J. V., and Weidman, F. D. Ibid., 1926, v. 13, May, p. 675). More recently Bloom and Mendelsohn have discussed the status of this form of cutaneous tuberculosis in the Negro (Cutaneous tuberculosis in the Negro, a clinical entity. Arch. Dermat. and Syph., 1937, v. 36, Nov., p. 637). It appears to represent a distinctive immunologic reaction of the Negro to tuberculosis. The cutaneous lesions cannot be classified with other types of cutaneous tuberculosis. They are not typical sarcoid or typical lupus, but appear to be a combination of sarcoid, hematogenous lupus vulgaris, and lupus miliaris disseminatus. In their onset they resemble the sarcoid of Boeck. Later the following distinctive features differentiate it from sarcoid, as well as from other clinical types of tuberculosis: the frequent involvement of the scalp, the psoriasiform character of the lesion, the ulceration and tendency of the lesions to follow the lines of cleavage of the skin. The histologic picture unlike that of sarcoid is that of bacillary tuberculosis as it commonly affects the viscera.

As in this patient, ocular lesions of tuberculosis may be one of the first symptoms preceding cutaneous involvement. Other characteristic features of the disease are: At the onset of cutaneous involvement there is absence, or inconspicuous evidence, of pulmonary tuberculosis. The cutaneous tuberculin test is at first negative, later becoming positive. Roentgenogram of the long bones frequently shows characteristic changes. Guinea-pig inoculations of cutaneous lesions are negative despite the histologic picture of bacillary tuberculosis. Later there develops a frank pulmonary and visceral tuberculosis. At this stage the cutaneous lesion has undergone involution with scar formation. More women than men are affected.

Of interest to the ophthalmologist is the occurrence of ocular lesions preceding cutaneous involvement, the negative cutaneous tuberculin test, and the diagnostic evidence afforded by roentgenograms of the long bones.

The changes that occur in detached retinas

Dr. Algernon B. Reese (New York) said the changes that occur in simple serous detachment of the retina could be discussed under the following headings: 1. Cysts appear early and first in the external plexiform layer of the macula. These are degenerative manifestations due to a nutritional disturbance that affects the avascular macula first. They account for the poor central vision so frequently obtained in cases in which the retina has been successfully reattached by an operation. These cystic spaces later appear throughout the entire retina and

tend to become larger and larger. The large cysts protrude entirely from the external retinal surface into the subretinal space and therefore cannot usually be seen clinically. If there is a disinsertion at the ora serrata, so that one can look into the subretinal space, they may be seen clinically. If one operates on a detached retina with large cysts, no reattachment can be obtained, for only the cyst wall comes in apposition to the choroid. The cysts just described are all secondary to the detachment. Weve has described primary cysts in the periphery of the retina and claims that they may cause a disinsertion at the ora serrata and thus a detachment of the retina.

2. The white lines frequently seen coursing over a detached retina as it is viewed with the ophthalmoscope, are due to the friction of the rods and cones against the choroidal surface at the limit of the detached area. This friction causes the replacement of the rods and cones by opaque glial tissue. Further detachment of the retina may cause such friction lines to form at other sites.

3. The Scotch-grain-leather appearance frequently noted over the surface of a detached retina is due to wrinkling of the internal limiting membrane of the retina.

4. A retina that has been detached for several years undergoes a degeneration of its nuclear elements and may be reduced to a glial membrane. This contracts and shortens the retina.

5. The only observation in regard to the etiology of detachments that could be made from the histological study of 12 relatively early cases of simple serous detachment of the retina, was that three showed disinsertion at the ora serrata, four showed detachment of the vitreous, and one showed a hole that had apparently occurred from a low-grade choroiditis. It was demonstrated that hemorrhage or exudate in the posterior chamber of the eye may remain for a long time along the avascular anterior hyaloid membrane of the vitreous and ultimately become organized. This thin fibrous membrane, extending from the hyaloideocapsular ligament to the ora serrata, may contract and pull the retina forward. This type of detachment may follow a contusion of the eye or a transitory and even very mild iridocyclitis. It naturally offers an extremely poor operative prognosis.

6. An eye which has harbored a detachment of the retina for some time frequently develops a uveitis, maybe with secondary glaucoma. This seems to be due to the fact that the subretinal fluid, which becomes increasingly more toxic with time, escapes through some atrophic area in the retina and produces an inflammation.

Discussion. Dr. James S. Shipman said that Dr. Reese's microscopic sections made very plain the reason why operative treatment for retinal detachment so often fails.

His experience with retinal detachment has been more in a practical way, and in discussing Dr. Reese's paper he wished to give a brief report of the results which Dr. B. F. Baer, Jr., and he had obtained in a consecutive series of 70 cases in which one or the other or both of them had operated for retinal detachment. In 20 of these cases two operations were performed, and in three of them three operations. Retinal tears were seen in 50 percent of the total number of cases. In seven cases of aphakia only one was improved by operation. Out of the entire group there were 35 successful cases. Thirteen patients were improved by operation and 22 unimproved. Of the unimproved six were aphakic. In 19 of the successful cases the patients obtained 6/15 or better vision. Three obtained 6/21 vision. The 13 remaining patients obtained 6/60 to 6/30 vision. Three of these were

known to have been amblyopic before and after operation. After hearing Dr. Reese's discussion, he is sure the condition in these cases represented a cystic degeneration of the macula. In the remaining nine cases he believed vitreous opacities from hemorrhages or too much diathermy accounted for poor vision in some, and high myopia with retinal degeneration and the presence of the detachment for a long time accounted for others. Myopia was present in varying degrees in approximately 40 to 50 percent of the cases. The youngest patient operated on was 15 years old and the eldest 79 years; in both the operation was a failure. There was a history of trauma in 18 cases, eight of which were successfully treated. No detachment recurred after replacement had held for three months or more. A great majority of the successful cases reported have stayed so for one year or more. Some have been holding for as long as five years.

In any discussion of retinal detachment the causes of this disturbing condition are always a question of debate. Arruga says "The role of the retinal holes in the development of a detachment is incontestable, but the difficulty is to interpret how these perforations act." Arruga has also demonstrated in rabbits that chemical or physical irritation to the sclera externally may give rise to a reaction in the choroid and the retina beneath as well as in the underlying vitreous, causing a detachment of the retina in one of two ways; either by contraction of the vitreous, drawing the retina away from the choroid, or by a swelling of the choroid with exudate pushing the retina away from it. Retinal degeneration in myopic eyes, which may be cystic, sometimes is an underlying cause. A previous uveitis or choroiditis with a breaking-up and contraction of the vitreous structure may well be a predisposing factor in retinal tears or detachments. Physical trauma in a mild degree, in an unhealthy or predisposed eye, or of a severe degree in a healthy eye, must still be recognized as a definite cause of detachment.

Dr. Shipman was impressed with the slide which Dr. Reese showed of a solitary cyst near the ora serrata which was giving rise to a retinal detachment. This certainly explains the futility of trying to get the retina back in place. Dr. Peter Kronfeld of Chicago feels that solitary cysts of this type are the most frequent cause of detachment in children and young adults. He has many times seen the white lines in the retina which Dr. Reese so beautifully demonstrated, and he felt that the explanation of them is very logical, namely friction lines.

The operative results for retinal detachment are certainly unpredictable. The most favorable cases seem to be disinsertions of the retina from the ora serrata, if not too large and if not caused by a solitary cyst; flat detachments of recent occurrence; other detachments with small or few holes fairly well forward. The group of unfavorable cases include extensive and complete bullous detachment of long standing; large detachments which do not tend to go back when the patient is put to bed; any detachment involving the macula; all detachments with cloudy media; soft eyes resulting from previous uveitis; degenerated or atrophic condition of the retina so often seen in highly myopic eyes, which are usually due to a cystic degeneration.

In conclusion he believes that operation for retinal detachment is only palliative and not a permanent cure, just as operation is for glaucoma. The cause of retinal detachment must first be removed to secure a permanent cure, and to do this we must yet learn more about the causes.

Warren S. Reees,
Clerk.

MINNESOTA ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

Section on Ophthalmology

January 21, 1938

Dr. Walter E. Camp, *president*

Detachment of the retina

Dr. John P. Macnie of New York City presented slides and moving pictures.

Discussion. Dr. Erling Hansen was a little surprised when Dr. Macnie said that in their experience the statistics showed that the length of time the detachment had been present before operation did not make much difference.

Dr. Macnie said that detachments which are a result of pregnancy have a good prognosis without treatment, but the detachment may merely be coincident with the pregnancy, and this type has been operated upon successfully. As to the duration of the detachment not seeming to influence the result, they were very much surprised to find this in analyzing the results of their cases. They had some cases of three or four years' duration that were restored with function.

While they do not like to see the retina out of position when the first observation is made after operation, this does not necessarily mean failure. They like to keep the patient in bed for a period of two-and-one-half weeks, and if there is no improvement then the patient is allowed to be up.

The eye does become soft during the operation, but this does not interfere. Penetration takes place with very gentle pressure. If the end of the wire does not go through the sclera very easily, they increase the strength of the current.

George McGeary,
Secretary.

CHICAGO OPHTHALMOLOGICAL SOCIETY

January 17, 1938

Dr. Thomas D. Allen, *president*

Aneurysma racimosa arterio-venosum

Dr. J. C. Dunbar said that this boy, six years of age, of Italian extraction, was seen at the Illinois Eye and Ear Infirmary with the complaint that since the age of three years his left eye had "turned out." Subsequent history disclosed that on several occasions when the patient had "fever" the left side of the face became flushed. Vision R.E. 20/20, L.E., no light perception. It was found that the patient was atropine sensitive. A 20-degree divergent squint was manifest. Ophthalmoscopically the right fundus was normal. On examination of the left fundus many excessively large and tortuous vessels were conspicuous, having the appearance of a bag of angleworms. The arteries could not be differentiated from veins, and numerous capillary anastomoses connected the vessels. There was gradual tapering of the vessels as they passed anteriorly. The retina, although nearly obscured by the large vessels, everywhere appeared normal. No angiomatous masses of glial proliferation were evident. The difference in temperature on each side of the face was noticeable.

Transillumination showed conspicuous vessels in the left cheek and ear, whereas they were barely recognizable on the right side of the face. A clinical thermometer placed beneath the tongue on the left recorded one degree higher than on the right. Blood pressure was equal in both arms. Neurologic examination was negative. There was no history of convulsions, syncope, or symptoms referable to the nervous system. The boy was very cooperative in all respects, and appeared above average intelligence. Pediatric and

nose and throat consultations added no further findings. All laboratory tests were normal.

The condition was considered to be a congenital vascular anomaly, possibly an atypical case of the Von Hippel-Lindau syndrome. Dr. von der Heydt photographed the fundus and added that similar cases have been described in the foreign literature as aneurysma racimosa arteriovenosum. He further stated that he could find only nine similar cases reported up until 1933.

Staphylococcus conjunctivitis

Drs. C. S. O'Brien and J. H. Allen read a paper on this subject which was published in this Journal (June, 1938).

Discussion. Dr. J. H. Allen, in reply to Dr. Gifford, said that practically all these cases had come from Iowa. Dr. Thygeson had recently reported his observations made in Iowa City before July, 1936, and, in addition, a series of over 100 cases of staphylococcic conjunctivitis which he had observed since then in New York City.

In reply to Dr. Henry, he said that autogenous vaccine had been used alone in some cases; toxoid had been used alone in others; and in still others both were used. In the severe cases no remarkable difference had been observed. The best results, however, had been obtained with autogenous vaccine alone in cases of chronic blepharitis without an associated conjunctivitis and in cases of repeated styes in which there was no other ocular disorder nor associated general physical disturbance. The dosage of autogenous vaccine was generally started with 0.1 c.c. and increased by 0.1 c.c. at each succeeding weekly injection until a dose of 1.0 c.c. was given. Then the 1.0-c.c. injection was repeated at weekly intervals until approximately 10 c.c. of the autogenous vaccine had been given.

The vaccine contained approximately one billion dead staphylococci per cubic centimeter and was prepared as follows: After having been isolated in pure culture upon blood agar the organisms were grown for 24 hours upon plain agar. A heavy suspension of the growth was made in saline and placed in a constant-temperature waterbath for one hour at 60°C. Subcultures were made on three successive days to make certain that the suspension contained no viable organisms. Saline was added until the suspension just matched the No. 3 barium-sulfate nephelometer standard.

The age range in the most severe cases, of which 39 had been observed in the past 18 months, was from 12 to 59 years, with the majority occurring between the ages of 30 and 40 years.

With a reference to treatment of corneal ulcers in staphylococcic conjunctivitis, various methods were used, but apparently the best results had been obtained in the four cases treated by staphylococcus antitoxin. The eyes were cleansed by irrigation with sterile saline twice a day, and 0.2-percent scopolamine solution was instilled three times a day. Each patient was given 40,000 units of the antitoxin by intramuscular injection.

Dr. Gifford had asked whether organisms were found in the ulcer. In one recent case a smear was made direct from the ulcer and a number of staphylococci were found. Before Dr. Thygeson left Iowa City, he demonstrated staphylococci direct from ulcers by smear and culture in several cases. Dr. Brown of Cincinnati was able to produce rather persistent ulcers in rabbits by the inoculation of staphylococci into the corneal stroma. In their own work, deep epithelial lesions had been produced in the corneae of rabbits and monkeys by the repeated instillation of staphylococcus toxin.

Someone had mentioned the possible

relationship to infection with Morax-Axenfeld bacilli. Because these cases presented angular excoriation, slight conjunctival discharge, and more intense inflammatory reaction of the caruncle and lower cul-de-sac in addition to blepharitis, it was realized that H. lacunatus must be considered as a possible etiologic agent. The absence of these organisms was demonstrated in all the cases presented as staphylococcic conjunctivitis by scrapings from the lid margins as well as the conjunctiva, and by cultures taken upon chocolate agar and Loeffler's slants in addition to the routine blood-agar plate. Apparently the Morax-Axenfeld bacillus is not very common in Iowa, for only 13 cases were seen in which that organism occurred alone, and three in which it occurred with other organisms in the series of 2,110 cases of conjunctival inflammation.

In reply to Dr. Halper, he said that no correlation studies had been made between the occurrence of staphylococci in the eye and in the nose.

Dr. Woodruff had asked about treatment with zinc sulfate. Strong solutions had not been used, as recommended by some for the treatment of acute conjunctivitis, but 0.25- and 0.5-percent solutions had been used in the chronic phases. No difference, however, had been observed in the course of patients using this drug and those using oxycyanide of mercury or phenyl mercuric nitrate. Quinine bisulfate had not been used in any cases.

With reference to treatment of the tear sac, in all these cases the nasolacrimal ducts were irrigated at least once to determine their patency. The cases of staphylococcic lacrimal conjunctivitis were treated in the same manner as other types of dacryocystitis. The sacs were irrigated often enough to keep them clean, and if they did not respond, probing or surgery was resorted to.

Dr. C. S. O'Brien, in reply to Dr. von der Heydt's question concerning the number and kind of floaters, said that one sees approximately two or three times as many cells floating in the aqueous in a patient 25 to 30 years of age as would be found in the senile type of nonpathologic eye. All these patients were examined with the slitlamp, and this was just an observation. He did not know the type of cell, but thought that they must be an indication of a mild type of iritis or irritation, perhaps as a result of the corneal condition, although they did not depend upon the presence of ulceration. He had not seen them with other cases of conjunctivitis.

In reply to Dr. Mayer's question, he said that no eye was operated on from which pathogenic microörganisms of any type were cultured. This applied to hemolytic staphylococci, Diplococcus pneumoniae, and any other type of organism capable of producing ocular disease. The patients were sent home under treatment, or were treated in the clinic; but if the cultures continued to show growth, no operation was performed.

Robert von der Heydt.

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RETINAL ANGIOMATOSIS AND GLOMUS TUMORS

Probably the first recognized case of retinal angiomatosis was reported by Ernst Fuchs in 1888. In 1892 D. J. Woods saw a case in the service of Mr. Tweedy. He made, in colors, a sketch of the fundus of one eye, which was presented at a meeting of the Ophthalmological Society of the United Kingdom and published in the Transactions of the Society. The patient was a young man of 20, whose other eye was already blind. An older sister had become blind in both eyes. In this condition she had come to Mr. Morton. The eyes were excised and given to Mr. E. Treacher Collins, who also had the excised eye of Mr. Wood's patient. In 1894 Mr. Collins reported upon the anatomical conditions found in these cases. In 1895 Dr. Wm. C. Bane, of Denver, saw a case, of which he made an accurate pen-and-ink drawing. He later exhibited this at a meeting of the Colorado Ophthalmological Society, where it was recognized as a case of retinal angiomatosis, as described by Mr. Collins. In 1896 von Hippel encountered a case, which he described and showed from time to time at the German medical societies. After the death of the patient, in 1903, the eye was examined and reported upon in Graefe's Archiv. The condition has been known in Germany as von Hippel's disease.

As pointed out by Collins, the essential abnormality is an arterio-venous anastomosis. It usually occurs in both eyes and has been recognized in two or more members of a family. In 1926 Lindau pointed

* Deceased.

out that it was sometimes associated with what he called cystic tumors of the cerebellum. But these tumors resembled the lesions of retinal angiomatosis.

Glomus, so called from its resemblance to a ball of yarn, is a form of nerve ending, found normally in the deeper layers of the skin, particularly in the palms of the hands, the soles of the feet, and most numerous in the matrix of the nails. It is a fraction of a millimeter in diameter and consists chiefly of nerve fibers interwoven with minute blood vessels making an arterio-venous anastomosis. The glomerulus of the kidney is named for a similar resemblance to a ball of yarn. The glomus tumor is a mass of small arteries surrounded by nerve fibers. Such tumors are likely to become painful; on that account, they have often been called painful tubercles, and excised. But their real nature and significance were not recognized. They have been classed as angiosarcomata, or peritheliomata.

In 1924 Masson recognized their resemblance to, and connection with, the "neuro-myo-arterial glomera" of the normal body. Since he called attention to glomus tumors, 65 cases have been reported. But they have generally been regarded as rare. Pain, the most prominent symptom, has been noticed, often for many years, before the patient applied for relief. In the Lancet for May 14, 1938, Kendall and Thomson have reported three cases observed in one hospital within two years. One patient had known of his tumor for 10 years; another had noticed the pain under her thumbnail, intermittently, for 12 years. The other had suffered pain under her thumbnail for five years, and twice had the nail been removed.

It can hardly be doubted that these tumors formed by the overgrowth of minute arterio-venous anastomoses, are essentially similar in origin and character to the lesions of retinal angiomatosis. The early impairment in vision in retinal angiomatosis probably forces the disease upon the attention of the patient earlier than it would be recognized by pain in other parts of the body. Although usually pain from increased intraocular tension ultimately compels the enucleation of the blinded eye, it would be interesting to observe the effects of early lesions in the retina, or their surgical destruction, upon the changes going on in other parts of the retina, or on the retina of the fellow eye. The disastrous course of such lesions, left undisturbed by surgical interference, will quite justify radical treatment as soon as the diagnosis can be established.

Edward Jackson.

MYOPIA AND BODILY STRUCTURE

The relative parts played by heredity and environment in the development of myopia were discussed in these columns a few months ago (American Journal of Ophthalamology, 1938, volume 21, page 680) in relation to a monograph by Stig Holm which reported special investigation among primitive negroes of tropical Africa. The Swedish author, like Cohn, Stilling, Steiger, and earlier workers, leaned strongly to the opinion that the tendency to myopia (or to other marked refractive errors) was hereditary or racial.

In the past few years there has been a noticeable trend away from the belief that mere use of the eyes must be blamed for the development of myopia during school age. The years of attendance at school are years of growth, years in which dominant hereditary influences are likely to express themselves through rapid variations in bodily structure.

The problem of myopia continues to lend itself much better to speculation than to the experimental method. However carefully we assemble facts which seem to bear upon the problem, the results are never so final as to exclude opposing views, and we are dealing at best in more or less weighty probabilities.

Such a criticism may be applied rather easily to the attempt by Francke (Klinische Monatsblätter für Augenheilkunde, 1938, volume 101, page 184) to prove that the development of myopia is closely related to bodily structure and growth. But the facts which that author has assembled will at least add to our ability to form a balanced judgment.

In 1924 Vogt traced a relationship between the size of the retina and the volume of the posterior segment of the eyeball. According to his conclusions, the inheritance of a large retina would give rise to a long ocular axis and therefore to myopia, while hereditary transmission of a small retina would result in a short axis such as is found in hyperopia.

In 1928 Incze reported his findings in 873 patients of an eye clinic with regard to the relation between refraction and bodily type. He found evidence that hyperopia was characteristic of a stocky build and myopia of an asthenic build. But the foundation upon which these conclusions are reached is none too secure. The myopic subjects were classified as being 75 percent asthenics and 20 percent of the robust type, while the hyperopic were divided into 39 percent of the stocky type and 52 percent asthenics. A later investigation by the same author placed the asthenic percentage among the myopic at 72.3 percent. The investigations were based upon external characteristics and not upon anthropometric data.

Francke's present report on the problem was conducted in Berlin clinics and took into consideration differences of general physique, of height, of body structure, and of cranial formation in the individual refractive groups; using measurements of size, weight, sitting height, length and breadth of head, width of forehead, pupillary distance, and the protrusion of the corneal apex beyond the line joining the external angles of the orbits (exophthalmos). The patients were classified as leptosomatics (slender), pykniks (stocky), and of medium growth.

Francke's general conclusions support those of Incze. Among the myopic cases leptosomatics predominated, whereas among the hyperopic a pyknik build was more frequent. The bodily length of the myopic persons averaged about one and one-third inches more than that of the hyperopic. However, in cases of high myopia, such as are encountered in very early childhood, there was a fall in the averages of bodily size, and these cases included subnormal or even "dysplastic" types.

It is an interesting fact that in a single family a hereditary tendency to marked variation from emmetropia sometimes seems to express itself in different ways. Thus one child will be decidedly hyperopic, another conspicuously myopic, and a third strikingly astigmatic. This may of course be due to mixed hereditary influences; but may it not be, on the other hand, in some instances merely an expression of familial flexibility of type, the progressivism which was referred to in dealing with Stig Holm's monograph?

Most of us may not find it easy to arrive at dogmatic conclusions on this subject, but the facts presented by Vogt, Incze, Francke, Sorsby and others seem to lean in the direction of heredity and physical constitution as the predominant influences in the development of myopia.

W. H. Crisp.

SOUTHERN MEDICAL MEETING

This was the editor's first visit to Oklahoma City. It looks like the "Spotless Town" from which Sunny Jim Force used to deliver his homilies. After an overnight trip on an exceptionally good train—and they are really making them now—from the soft-coal belt, this city looked as if it had just emerged from a band box. Many of the large buildings are new and shining white; very cozy and warm on a bright November day. If only natural gas were available at bargain prices for all of our big cities!

Our Section met in the new Municipal Auditorium. We were assigned a relatively small room in which a loud speaker was not necessary. This has certain psychological advantages for our Section; such a room is more apt to be well filled. That in itself is stimulating. We like to go where the other fellow goes. A group that fills a small room is easier to address than the same group scattered over a larger one. The microphone nuisance is avoided. Admitting the unquestioned advantage and even necessity of these instruments in large halls, they undoubtedly handicap the speaker's style and actions. It is surprising that the type of phone that can be pinned to the lapel of the speaker is not more frequently used. With the floor type the lecturer frequently varies his distance from the instrument, thus inappropriately modifying the resonance of his voice and even occasionally completely losing the instrument when he turns to point out some feature on the screen. The small room adds also to the intimacy, takes away the formality of the meeting, and lends itself better to discussion. Don't let me give the impression that the Section on Ophthalmology and Otolaryngology was not well attended. From 100 to 150 were present during both days that were given over to ophthalmology.

The Southern Medical Association has a charm all its own. It doesn't bristle with Big Business like the Academy; nor is it like the A.M.A., the greatest show on earth, Main Street come to Main Street. No, it is just a friendly get-together of folks from home. In our Section, certainly, discussion is free and easy; perhaps not all so well worth while as more formal, prepared discussions, there being sometimes a little too much of the "I recall a case" type; but, after all, men attend meetings for many different things, and one of them is to get up in meeting and talk. Some very good material leaks out that way too.

I dwell a little on this matter of free discussion because the lack of it is a problem in all of our large societies. At these meetings the discussor sends in later such part of his discussion as he desires to have printed. Probably some of it is forgotten and laziness eliminates more; hence considerable printer's ink is saved.

Although the Section on Ophthalmology and Otolaryngology has not seen fit to divide into two groups, largely because most of the members practice both specialties, there is an attempt to concentrate ophthalmological and otolaryngological subjects on different days for the convenience of those who specialize more strictly. The writer regretted his inability to attend both ophthalmological programs.

The ophthalmological section got off to a fine start with a splendid paper by Bernard Samuels, the guest speaker, on the pathology of various types of cataract with the practical implications for their clinical handling. The talk was illustrated with beautiful slides. Among the many interesting comments, perhaps those associated with cataracts secondary to inflammations were especially good. The speaker advised waiting for at least a year after subsidence of a chronic uveitis before operating for the cataract, and always a preliminary iridectomy. He ex-

plained the failures in many instances of needlings as due to damage to the anterior face of the fluid vitreous. He advised against extraction of the pearly cataract following retinal detachment, although in discussion it was brought out by one surgeon that he had had excellent success with intracapsular extraction in these cases. This paper was an outstanding contribution and should be read at leisure. It will appear in the Southern Medical Journal.

A long and interesting discussion on treatment of trachoma followed. The value of sulfanilamide in this condition was stressed by the discussors, although it was not discussed by the essayist. To one who has been an observer in the thick of the thrilling advances in the study of the etiology and treatment of this disease in the past few years it is surprising to hear those who have been quite uninfluenced by these discoveries.

Retinal detachment was the subject of two of the ten papers. Heat therapy was considered at some length, and, briefly, the value of ultraviolet light as a fluorescence-producing agent was illustrated with motion pictures.

We are always seeking hidden causes for asthenopia. Latent phorias explain some of these cases, aniseikonia others. One essayist discussed very interestingly his observations on some 150 patients with especial reference to hypothyroidism. Among principal signs and symptoms were cited peripheral constriction of the fields, particularly to green and red, and lowered basal metabolic rate. Fields showed variations directly with changes in this rate. These observations are certainly worth following further.

Memphis was selected for the next meeting place and the following officers were chosen: president, Walter E. Vest, Huntington, Virginia; president-elect, Arthur T. McCormack, Louisville, Kentucky; chairman of the Section of Ophthalmology and Otolaryngology, Lawrence T. Post, Saint Louis, Missouri; vice-chairman, Harvey B. Searcy, Tuscaloosa, Alabama; and secretary, Lawrence Chester McHenry, Oklahoma City, Oklahoma.

Lawrence T. Post.

BOOK NOTICES

PATHOLOGIC ANATOMY AND THERAPY OF TRACHOMA AS FOUND IN THE TARSUS AND ITS CONJUNCTIVA. By A. Birch-Hirschfeld. Paper covered, 42 pages, 18 illustrations. Supplement No. 27 to Zeitschrift für Augenheilkunde. Basel and Leipzig, S. Karger, 1938.

After more than 20 years of histologic examination of a very large number of tarsi, removed because of trachoma, together with the clinical observation of trachoma cases in a district where the disease is prevalent, Birch-Hirschfeld is convinced that the pathology of trachoma is not the simple, easily comprehensible condition the text-books and hand-books describe. After such an experience as Birch-Hirschfeld's, any observer would be astonished by its multiplicity of disease-forms, both clinical and pathologico-anatomical. Then one would understand that a schematic division and superficial review of the principal changes is not justified by the facts. On closer examination, an entire series of questions obtrude that have not been solved and for whose solution it is important to strive.

From the histologic examination of 153 excised tarsi with their covering conjunctivae, necessarily removed in the later stages of trachoma, the author comes to some definite conclusions. If the disease is divided into stages, as is done by McCallan, it must be understood that

these stages very often mingle in a single case and by no means follow one another in a definite order. In the overwhelming majority of the author's cases, cellular infiltration is still observed in the subepithelial tissue, pretarsal tissue, and in the tarsus, together with advanced degenerative and cicatricial changes. The formation of connective tissue, arising principally from the adventitia of the blood vessels, begins early in the foci of cellular infiltration. In this study, therefore, Birch-Hirschfeld does not divide the pathologic changes into stages of the disease but into those affecting the four following layers: 1, the epithelium; 2, the subepithelial tissue; 3, the pretarsal tissue; and 4, the tarsus.

The essential pathologic finding in trachoma is not the follicle but the inflammatory changes which lead to infiltration and loosening of the tissues, to necrosis and the proliferation of fibroblasts, in the subepithelial and pretarsal tissues and in the tarsus. In only 12 of the 153 excised tarsi was the tarsus unaffected, while in the subepithelial and pretarsal tissues the tarsal conjunctiva presented intensive infiltration and cicatricial changes.

In the treatment of trachoma, Birch-Hirschfeld employs expression or rolling, rubbing with sublimate solution on cotton-swabs, and silver nitrate, targesin, or chaulmoogra oil with preparations of copper in the case of papillary hypertrophy. When the tarsus is involved, nothing but its excision with the covering conjunctiva is effectual. This operation of Kuhnt and Heisrath has proved over many years of its performance to be of the greatest benefit and to be unattended by any complication.

Although much of what the author says is not new, nevertheless the work is valuable because it comes from the matured and experienced observation and judgment of a great eye-clinician and eye-pathologist.

L. A. Julianelle.

LA TONOSCOPIA RETINICA. By Dr. F. Orzalesi and Dr. N. Cassuto. Paper covers, 186 pages, and 8 plates. Florence, R. Bemporad & Figlio, 1938.

This book deals with tonometry of the retina and brings together the available literature on the subject. Following upon a short historical sketch of the subject, the authors describe the different apparatuses devised for measuring the pressure of retinal blood vessels and incorporate directions for their use. The different instruments described are those of Bailliart, Bliedung, Seidel, Abramowicz, Sobanski, Uyemura and Suganuma, Kukán, and Keil. A tabulated analysis of various reports on the pressure of the retinal artery indicates, with a few exceptions, rather remarkably close determinations made by the different methods, the discrepancies perhaps ascribable as much to the operator as to the apparatus. The diastolic pressure is as a rule close to 30 to 35 mm. Hg and the systolic 60 to 70 mm. and, as one might expect, there is a gradual rise in pressure with advancing age. Determinations made on the elasticity of the walls of the central artery of the retina reveal that under normal conditions elasticity varies directly with pressure and inversely with caliber.

A short chapter is devoted to the pressure of the retinal capillaries, with the conclusion that at the present time the methods devised for this purpose are unsatisfactory and uncertain. The venous pressure is measurable by different methods, with the apparatus of Sobanski giving accurate results, these showing values of 28 mm. Hg for the ages of 7 to 15 years, and a minimum of 16 to 22 mm.;

from 15 to 40 years, maximum pressure is 34 and minimum pressure 20 mm.; beyond 40 years, the variation is 26 and 23 mm., respectively. The relation between venous diastolic pressure and arterial diastolic pressure shows a proportion varying from 1:1.9 to 1:1.3, respectively. From the venous pressure, it becomes possible to determine blood flow by Fritz's formula.

The pressure of the retinal vessels may be influenced by various physiological and nervous conditions, as, for example, general hyper- or hypotension, pain menstruation, pregnancy, and, according to some authors, even by a change of posture. So also changes in pressure may be obtained by stimulation of the sympathetic nervous system. The effect of different drugs on pressure varies with the drugs: adrenalin alters the retinal pressure similarly to the general pressure, an effect apparently neutralized by cocaine; cocaine first raises and then lowers the pressure, as does also eserine; pilocarpine decreases while cobra venom increases pressure: acetylcholine induces a drop in pressure with a dilation of the right cerebral artery; and several other drugs are without effect or of indifferent result. Apparently, however, there is frequent discord among different workers on the changes caused by different drugs, and this phase of the subject requires further study.

The variations found in general blood pressure have been studied in relation to the pressure of retinal vessels. With certain exceptions, general changes are reflected by retinal changes. Increased pressure of the spinal fluid is usually associated with increased pressure of the retinal vessels. Stimulation of nerves in the neighborhood of the eye usually creates an increase in pressure of the vessels of the retina. A general impression obtained from this book is that the subject is in need of development before its true value in ophthalomolgy may be ascertained. Present knowledge indicates numerous contradictory observations, which the future will have to appraise as suggesting either unfamiliarity with methods and techniques, or as insignificant or indifferent effects. L. A. Julianelle.

REFRACTION OF THE EYE. By Alfred Cowan, M.D., Associate Professor of Ophthalmology, Graduate School of Medicine, University of Pennsylvania. Clothbound, 319 pages, 3 colored plates, 172 engravings. Philadelphia, Lea and Febiger, 1938. Price $4.50.

In his preface the author states: "For a long time it has been my desire to write a book on clinical refraction employing the theory of ophthalmic optics in such a way that clinical aspects would emerge logically in orderly sequence from their bases of scientific facts." This book is the working out of that purpose. When John Green, Sr., A.B., A.M., M.D., of Harvard University, after teaching for 10 years as Professor of Ophthalmology in the Saint Louis Medical College, found it was difficult to understand papers on mathematical optics, he engaged teachers of higher mathematics who gave him private lessons, and spent much of his time for two or three years in that study. After that he wrote the best paper to be found in the literature dealing with the refraction of oblique pencils of light.

When Helmholtz published the first part of his "Physiological optics," he explained why mathematical formulas were needed in a book devoted to the physiology of vision. Donders, in his "Accommodation and refraction of the eye," said the mathematical portions of his book had been approved by his colleague, Professor Hoek, the astronomer. The prelimi-

nary requirements for the study and practice of medicine usually include very little mathematics, and no optics. Physicians who need to understand mathematical optics, to deal with problems of refraction, have found little help from the available books on the subject.

Dr. Cowan's book will help substantially to fill this gap in our literature. It is the best book we have to prepare for the authoritative works on Newton, Helmholtz, Sturm, Gullstrand, or Tscherning. Cowan gives algebraic formulas, by which mathematicians have solved optical problems. But he also gives diagrams that explain the formulas and enable the student to understand the things he wishes to learn. By the aid of this book, many will find the difficulties that defeated them were not inherent in the subject, but were in the methods the mathematicians took to explain it.

After four pages of introduction, the chapters take up: Reflection and refraction at plane surfaces and at spheric surfaces; Lenses, thick lenses, cylindric lenses, ophthalmic lenses in general, and neutralization of prisms and lenses; The normal static eye, visual acuity, aperture of the system, accommodation, ametropia, and accessory effects of the correcting lenses, followed by 40 pages on Methods for determination of the refraction of the eye, Management and optical treatment of ametropia, and a chapter on Contact glasses and telescopic spectacles.

A bibliography of nine pages and an index of seven pages complete the book, making it a help that has been much needed by students who wish to understand and master the subject of ocular refraction. The illustrations are clear and properly placed. They are more numerous than would be supposed, from the list of them. For instance, Fig. 63, Focal intercepts, gives six figures; Fig. 7, Panoptic bifocals, gives seven different lenses; Fig. 16, gives 19 shapes and cross-sections of one-piece and fused bifocals. The colored plates illustrate the paths of different refracted rays. A few of the illustrations show ophthalmological apparatus. But these do not suggest that machines may be a substitute for professional experience and intelligent thought. Not all the needs of the ophthalmologist may be met by Dr. Cowan's book. But he who carefully studies it will be likely to get a broader view of mathematical optics and a better theoretic basis for methods of correcting optical defects of the eye.

Edward Jackson.

TRANSACTIONS OF THE AMERICAN OPHTHALMOLOGICAL SOCIETY. Volume XXXV. Clothbound, 431 pages. Philadelphia, Wm. F. Fell Company, 1937.

The seventy-third annual meeting of the American Ophthalmological Society was held in Hot Springs, Virginia, on June 3, 4, and 5, 1937. Twenty-eight papers, and a number of new instruments and appliances were presented. Twelve articles dealt primarily with intraocular pathology; two with retinal detachments, particularly with the results of surgical therapy; four were concerned with the extrinsic ocular muscles, and two with the physiology of the intraocular fluids.

Reese presents six cases of precancerous melanosis and diffuse malignant melanoma of the conjunctiva. He recommends exenteration rather than enucleation or local excision of the lesion. In the discussion of this paper the successful use of the thermophore was reported by Shahan, May, Lancaster, and John Green. Jackson states that eyestrain is the most important cause of cataract; in his opinion proper water intake along with the normal elimination of waste products will check the development of lenticular opacities. Barkan and Gray give an extensive

review of diabetes and diabetic retinitis. They urge the adoption of a uniform method of classifying the various lesions. Dunnington and Macnie report the operative results in 164 unselected cases of retinal detachment. A cure was obtained in 64 percent with less than one half of the retina detached; in 11 percent with three quarters or more of the retina detached. In the entire series cures were obtained in 42.1 percent.

Prangen and Koch report 54 cases of esophoria which they considered as divergence insufficiency. The use of prisms base out gave some comfort. Friedenwald and Stiehler present experimental work on the formation of the aqueous. Clay and Baird find that thyroparathyroid deficiency causes vascular and ocular hypotony. In their opinion ocular hypotony is an essential disease resulting in nutritional changes that are injurious. Lloyd reports six cases of birth injuries causing tears in Descemet's membrane and glass-membrane formations in the anterior chamber. Beach and McAdams use homatropine and benzedrine as a quick, effective, and relatively short-acting cycloplegic. Woods and Randolph report the therapeutic results of 175 cases of ocular tuberculosis. They conclude that small desensitizing doses of tuberculin given over a long period of time offer the most effective form of treatment. Verhoeff and Gross report three cases of disciform degeneration of the macula which follows hemorrhage between the pigment epithelium and Bruch's membrane. The appearance of a dark elevation in the macula is suggestive of a melanoma of the choroid. Differential diagnostic points are considered. An interesting case of an epithelial implantation cyst of the posterior chamber was shown by King. Vail reports the successful use of posterior sclerotomy in cases of expulsive choroidal hemorrhage. Useful vision was obtained in two cases. Castroviejo presents the microscopic findings in a large number of corneal transplants. Factors causing the success or failure of keratoplasty are enumerated. Cushman finds visual-field contractions and enlargement of the blind spots occurring in asthenopia associated with premenstrual headaches. Glandular therapy has been effective. A micro-camera and pictures of diseased conjunctival vessels were shown by Ruedemann.

This volume is of extreme interest and indicates the increasing interest in the basic sciences in the field of ophthalmology. William M. James.

CORRESPONDENCE

Contact Lenses

August 12, 1938
London

Dear Dr. Crisp:

I was interested to read your editorial on Contact Lenses in the July (1938) number of the American Journal of Ophthalmology.

For fairness to the Dallos technique I would like to say that Dr. Dallos (Budapest) has been working in London, at our Contact Lens Center in Cavendish Square, for rather more than a year, and during that time has made contact lenses for somewhat over 200 cases. He has used his own technique, making a cast of the eye and from this a contact lens for *each individual patient*—who is under the supervision of the oculist who has sent the case to the Center.

I have watched Dallos at his work and although I have never seen a Müller Welt type of contact lens, I cannot imagine a more perfect fit between the contact lens and the sclera than those I have seen finished by Dallos.

Yours sincerely,
(Signed) Margaret Dobson.

ABSTRACT DEPARTMENT

EDITED BY DR. WILLIAM H. CRISP

Abstracts are classified under the divisions listed below, which broadly correspond to those formerly used in the Ophthalmic Year Book. It must be remembered that any given paper may belong to several divisions of ophthalmology, although here it is only mentioned in one. Not all of the headings will necessarily be found in any one issue of the Journal.

CLASSIFICATION

1. General methods of diagnosis
2. Therapeutics and operations
3. Physiologic optics, refraction, and color vision
4. Ocular movements
5. Conjunctiva
6. Cornea and sclera
7. Uveal tract, sympathetic disease, and aqueous humor
8. Glaucoma and ocular tension
9. Crystalline lens
10. Retina and vitreous
11. Optic nerve and toxic amblyopias
12. Visual tracts and centers
13. Eyeball and orbit
14. Eyelids and lacrimal apparatus
15. Tumors
16. Injuries
17. Systemic diseases and parasites
18. Hygiene, sociology, education, and history
19. Anatomy, embryology, and comparative ophthalmology

1

GENERAL METHODS OF DIAGNOSIS

Berens, Conrad. **A chart for testing the visual acuity of amblyopic children and adults.** Trans. Sec. on Ophth., Amer. Med. Assoc., 1937, 88th session, p. 257. (See Amer. Jour. Ophth., 1938, v. 21, April, p. 444.)

Berens, Conrad. **Stereoscopic cards in color for children:** series B. Trans. Sec. on Ophth., Amer. Med. Assoc., 1937, 88th session, p. 254. (See Amer. Jour. Ophth., 1938, v. 21, April, p. 444.)

Berezinskaja, D. I. **Diffusion of acid and alkalies into the anterior chamber.** Viestnik Opht., 1938, v. 12, pt. 3, p. 356.

A laboratory study on rabbits, the conclusions of which are as follows: Mineral acids in 1-percent or higher concentration do not diffuse through the cornea. Strong alkalies diffuse into the anterior chamber, the more rapidly the greater the concentration. Increase of temperature in the alkali increases diffusion. Sodium hydroxide raises the permeability of the tissues to fluorescein. Fluorescein and sodium hydroxide penetrate from the conjunctiva through the cornea into the anterior chamber. The diffusion of fluorescein and sodium hydroxide is lower than the diffusion of sodium hydroxide alone. A circumscribed burn of the conjunctiva away from the limbus is not accompanied by diffusion of the alkali into the anterior chamber. Biomicroscopy and staining of the tissues are of great value in investigation of corneal permeability.

Ray K. Daily.

Horváth, B., and Németh, L. **Testing the visual field with the luminous perimeter.** Klin. M. f. Augenh., 1938, v. 100, June, p. 909.

Examinations of the visual fields of thirty healthy persons with the luminous perimeter in the dark gave almost the same results as those made in diffuse daylight. The visual field in glaucoma was from 5 to 10 degrees narrower in the dark than in diffuse daylight and the contraction at the nasal side confirmed the diagnosis. In choked disc an enlargement of the blind spot

was found at a time when the ordinary perimeter did not show anything pathologic. In some cases of atrophy there was only a slight difference, but in cases due to lues, especially in tabes, the contraction was so great that even during the examination it suggested the etiology. Perhaps most important was the use of the luminous perimeter in detachment of the retina, where it indicated a great defect of the visual field in very recent cases in which the diagnosis with the ophthalmoscope was difficult. Exclusion of disturbing reflexes is a great advantage of the method. However, fixation is not controllable and the examination requires good intelligence.

C. Zimmermann.

Leydhecker, F. K. **Observations on the capillaries at the edge of the fingernails in eye patients.** Graefe's Arch., 1938, v. 139, pt. 1, pp. 97-104.

From the clinic at Halle, 1,000 patients were examined under the microscope, with a magnification of about 35 ×, as to the capillaries and blood stream in the edge of the fingernail. A little cedar oil spread on the latter produced a definite reduction of the refraction of light at the surface of the skin. Types of change particularly noted in the capillaries were vasomotor diathesis, Jaensch blocking, diapedesis, and narrow capillaries. Capillary changes were much more numerous in women and in patients over forty years of age. Slight changes in the capillaries were more frequent in myopic females. Glaucoma did not appear to have any influence. H. D. Lamb.

Peyret, J. A. **Pupillometry and anisocoria.** Arch. de Oft. de Buenos Aires, 1938, v. 13, Jan., p. 16.

A method of differentiating between physiologic and pathologic forms of pupillary inequality is outlined. Using weak solutions of cocaine, pilocarpine, and eserine, the average amount of dilatation and contraction of the pupil in normal individuals was determined. The anisocoria is physiologic when the two eyes react with equal intensity, within the limits accepted as normal, and to all solutions employed. It is pathologic when each pupil reacts in a different manner and the excursions are greater or less than the normal values. The importance of employing the pharmacodynamic method in disturbances of the sympathetic system is stressed. Notes of a number of clinical cases are appended.

Edward P. Burch.

Wassermann, I. A. **Fluctuations in the size of the blind spot in the morning and in the evening.** Viestnik Opht., 1938, v. 12, pt. 2, p. 229.

Studies of the blind spot show that it is 2 to 5 percent larger in the morning than in the evening. In glaucomatous patients the fluctuation is much greater and may reach 15 to 25 degrees. Therefore the author recommends that studies of the blind spot be made in the morning. (Illustrations.)

Ray K. Daily.

2

THERAPEUTICS AND OPERATIONS

Belskii, A. A., and Deshina, A. I. **Mud treatment in ocular diseases.** Viestnik Opht., 1938, v. 12, pt. 2, p. 192.

A comprehensive review of the literature on this form of therapy, as a report of its application in ophthalmology. The author found the application of heated mud directly to the eye beneficial in acute as well as chronic diseases. He ascribes the effect to local hy-

peremia. General mud baths act through their effect on the vegetative nervous system. Ray K. Daily.

Filatov, V. P. **Therapeutic tissue transplantation in ocular diseases.** Viestnik Opht., 1938, v. 12, pt. 2, p. 157.

Reports of a case of retinitis pigmentosa and a case of tuberculous uveitis treated by transplantation of cadaver choroid and retina, with favorable results. Ray K. Daily.

Garchovskaja, B. A. **Surgical treatment of ectatic post-operative scars with iris prolapse.** Viestnik Opht., 1938, v. 12, pt. 3, p. 365.

The author prefers cicatricectomy to conservative treatment. (Illustrations.)
Ray K. Daily.

Khluser, G. P. **Convocaine as a substitute for cocaine for superficial anesthesia.** Viestnik Opht., 1938, v. 12, pt. 3, p. 383.

An investigation into the clinical properties of the substance designed to replace cocaine in U.S.S.R. It is found to be as effective as cocaine, and its greater toxicity can be compensated for by using weaker concentrations.
Ray K. Daily.

Kogan, E. C. **The duration of the action of atropine sulphate.** Viestnik Opht., 1938, v. 12, pt. 2, p. 251.

A laboratory study on the action of weak atropine solutions, with candidates for the flying service as test subjects, leads to the following conclusions: The most practical solution is 1 to 5000, which gives maximum dilatation of the pupil in one hour, with return to normal on the second day. A 1 to 2000 solution acts more rapidly and thoroughly, but lasts not less than three days. Introduction of 1-percent pilocarpin ointment at the completion of the examination checks the action of the atropine. The full effect of weak solutions on accommodation is manifested only after three hours, and refraction should be tested three hours after atropine instillation.
Ray K. Daily.

Motegi, A. A. **A hydrostatic approach to the posterior chamber for diagnostic and therapeutic purposes.** Brit. Jour. Ophth., 1938, v. 22, Sept., pp. 543-552.

Here is presented a technique of "intraocular irrigation" used in three types of cases in which, in the experience of the author, examination and treatment involve special difficulty. A 0.9-percent salt solution or Ringer's solution at body temperature is run through an incised wound in the eyeball. The globular form of the eye is thus restored and observation of the interior is made possible. Through the clear saline the fundus is more easily seen than through the haze of turbid vitreous. Removal of a foreign body, and lavage of infective material from the posterior chamber, become possible in panophthalmitis. There is a possibility of treatment under direct vision. Experiments were conducted with rabbits, the methods used being described. The technique is fully delineated with case reports presented under the three classifications of the author's experience, namely: (1) patients with poor visual acuity whose fundi are invisible from changes in the anterior segment of the eye or elsewhere; (2) application of intraocular irrigation in panophthalmitis, and (3) application of this operation for removal of nonmagnetic foreign bodies from within the eyeball.

Pathologic changes in the eyeball are not occasioned by such intraocular irrigation. The method is said to afford a possible cure for panophthalmitis in the early stages. Conclusions summarize the findings under seven pertinent heads. (References.)

D. F. Harbridge.

Poliak, B. L. **The effect of diathermoionization on the normal and diseased eye.** Viestnik Opht., 1938, v. 12, pt. 3, p. 324.

A description of an apparatus which combines diathermic and galvanic currents, and a report of a laboratory and clinical investigation on its ability to force iodine into the ocular tissues. The report shows that this combined current can force penetration of iodine into the interior of the eyeball and the orbit of the rabbit. Clinically this form of therapy is found very effective for corneal opacities. Ray K. Daily.

Promtov, V. A. **Synthetic pilocarpin.** Viestnik Opht., 1938, v. 12, pt. 2, p. 248.

A laboratory study on the properties of synthetic pilocarpin manufactured in Russia, and designed to replace the imported drug. Ray K. Daily.

Samoilov, A. I. **The role of paraspecific therapy in ocular tuberculosis.** Viestnik Opht., 1938, v. 12, pt. 1, p. 27.

Through reports of cases Samoilov illustrates the effectiveness of calcium administration in the therapy of ocular tuberculosis. In metastatic tuberculosis calcium serves to desensitize individuals hypersensitive to tuberculin, so that specific therapy may be administered. In allergic tuberculous symptoms calcium in combination with supportive measures but without the use of tuberculin may be sufficient to control the disease. Ray K. Daily.

Volovich, D. S., and Greenglas, A. I. **Transfusion in ocular diseases.** Viestnik Opht., 1938, v. 12, pt. 2, p. 226.

The author obtained favorable results from transfusion in vitreous hemorrhages and resistant postoperative iridocyclitis. Ray K. Daily.

3

PHYSIOLOGIC OPTICS, REFRACTION, AND COLOR VISION

Berg, Fredrik. **The optical properties of iseikonic lenses.** Acta Ophth., 1938, v. 16, pts. 2-3, p. 228.

A mathematical explanation of the fact that the iseikonic lens, while varying the size of the retinal image, is optically afocal. Ray K. Daily.

Bielschowsky, Alfred. **Aniseikonia.** Acta Ophth., 1938, v. 16, pts 2-3, p. 188.

A comprehensive review of the work done by Adelbert Ames, Jr., and his associates. Bielschowsky is convinced that aniseikonia is a factor in the etiology of strabismus, that it handicaps binocular vision and encourages suppression, and that its correction is a definite factor in obtaining ideal therapeutic results. Ray K. Daily.

Chase, A. M., and Haig, C. **The absorption spectrum of visual purple.** Jour. Gen. Physiology, 1938, v. 21, March 20, p. 411.

A technical article in which the authors conclude that it is very likely that the classical absorption spectrum is that of the light-sensitive group only and that the absorption spectra of the purest unbleached visual purple solutions represent the molecule as a whole.

Ralph W. Danielson.

Cushman, Beulah. **Visual field studies in asthenopia.** Trans. Amer. Ophth. Soc., 1937, v. 35, p. 385.

Visual-field contractions and enlargement of the blind spots were found in patients complaining of asthenopia and suffering from premenstrual headache and fatigue. Both the field contractions and the asthenopia improved on administration of anterior pituitary lobe extract. Visual-field studies may aid in the interpretation of glandular dysfunction and should be made in asthenopia where correction of refractive errors does not relieve symptoms.

David O. Harrington.

Elsberg, C. A., and Spotnitz. **A theory of retino-cerebral function with formulas for threshold vision and light and dark adaptation at the fovea.** Amer. Jour. Physiology, 1938, v. 121, Feb., pp. 454-464.

Mathematical formulas are given for threshold vision and for light and dark adaptation at the fovea.

T. E. Sanders.

Hartline, H. K. **Response of single optic nerve fibers of vertebrate eye to illumination of the retina.** Amer. Jour. Physiology, 1938, v. 121, Feb., pp. 400-415.

Action potentials in single optic-nerve fibers of cold-blooded vertebrate eyes may be obtained from small intraocular bundles dissected from the surface of the retina. The recorded response is studied in detail as to type of response, intensity of stimulation, effect of dark adaptation, and the receptive area of the particular fiber.

T. E. Sanders.

Hopkins, G. W. **Reading defects in school children.** Rocky Mountain Med. Jour., 1938, v. 35, March, pp. 218-221.

The relationship of ocular dominance and left-handedness to reading difficulties is discussed. It is suggested that the cross-eyed child has a greater tendency to reading difficulty.

T. E. Sanders.

Kentgers, S. K. **The relation between pregnancy, disturbance in dark adaptation, and vitamin-A content of the blood.** Acta Ophth., 1938, v. 16, pts. 2-3, p. 332.

Studies of the threshold of perception and of the vitamin-A blood content of pregnant and non-pregnant women show that a normal vitamin-A blood content is found less frequently in pregnant women with normal adaptation than in non-pregnant women; and that diminished adaptation is associated with reduced vitamin-A content more frequently in pregnant than non-pregnant women. The data also indicate that a reduced vitamin-A blood content is more frequently found in pregnant women, and that the threshold of perception curve does not run parallel to the vitamin-A blood content. Since half of the pregnant women with normal adaptation had a reduced vitamin-A blood content, it is fair to conclude that dark adaptation does not run parallel to the vitamin-A blood content. Studies on post-partum cases, as well as studies on cases under treatment with vitamin A, confirm the author's conclusion. The administration of vitamin A improves dark adaptation, regardless of its effect on the vitamin-A blood content. This seems to indicate that the latter is not an indicator of saturation of the body with vitamin A. Disturbance in dark adaptation is not an early symptom of A-avitaminosis, and it is probable that adaptation disturbances take place after the organism has used up its store of vitamin A.

Ray K. Daily.

Kogan, E. C. **The duration of the action of atropine sulphate.** Viestnik Opht., 1938, v. 12, pt. 2, p. 251. (See Section 2, Therapeutics and operations.)

Krause, A. C., and Sidwell, A. E., Jr. **The absorption spectra of visual purple.** Amer. Jour. Physiology, 1938, v. 121, Jan., p. 215.

Fluid rich in visual purple and obtained from retinae ground in sodium-chloride solution was used for the experiments. When exposed to light a photochemical reaction occurred which resulted in the splitting of visual purple into protein and a colored lipid called visual yellow. Further exposure to light or heat changed the colored lipid into a colorless lipid. Evidently vitamin A was not formed by the photochemical reaction of visual purple or of visual yellow. F. M. Crage.

Kravkov, S. V. **The influence of dark adaptation on the critical frequency of flicker for monochromatic lights.** Acta Ophth., 1938, v. 16, pts. 2-3, p. 375.

The objective of this investigation was to determine the course of the critical frequency to monochromatic flicker stimuli during dark adaptation. The charted results show that the critical frequency falls with dark adaptation, with the greatest fall for blue stimuli, less for red and yellow, and practically no fall for green. A protanope thus tested showed a considerable fall in the critical frequency for blue stimuli, and a uniform rate of fall for the rays of all other wave lengths. The author interprets this finding as an indication that in the protanope the green and red sensitive apparatuses are not differentiated. The author attributes the fall in critical frequency during dark adaptation to a diminution in the contrast sensibility of the eye, a function probably controlled by higher visual centers. Ray K. Daily.

Kravkov, S. V. **Illumination and visual acuity.** Acta Ophth., 1938, v. 16, pts. 2-3, p. 385.

The objective of this investigation was to elucidate the factors which account for the rise in visual acuity with the increase in illumination. Wilcox attributes the phenomenon to the effect of irradiation, while Kravkov maintains, and his stand is supported by the results of this study, that an important factor in addition to irradiation is the contrast sensibility of the eye, which rises with illumination.

Ray K. Daily.

Lindahl, J. **Two cases of transitory myopia.** Acta Ophth., 1938, v. 16, pts. 2-3, p. 344.

A review of the literature and a report of two cases, in which the general examination indicated a disturbance in salt and water metabolism.

Ray K. Daily.

Musilev, F. I. **The functional dependence of the retinal periphery on its center.** Viestnik Opht., 1938, v. 12, pt. 2, p. 261.

A laboratory study on physiologic optics, the charted results of which lead to the following conclusions: Stimulation of the green-and-blue-perception elements in the parafoveal cones has a stimulating effect on functional restoration of the retinal periphery. This reciprocal stimulating action takes place not in the fovea, but in the green-blue-perception elements of the parafoveal cones on the investigated side.

Ray K. Daily.

Ohm, J. **Healing of nystagmus in two siblings with color blindness.** Zeit. f. Augenh., 1938, v. 95, July, p. 200.

Ohm reëxamined a pair of siblings with color blindness in whom he had recorded tracings of nystagmus in 1925 and 1928 respectively. When reëxamined in 1937 every vestige of nystagmus even in the end positions of ocular rotation had disappeared.

F. Herbert Haessler.

Riddell, W. J. B. **A hemophilic and color blind pedigree.** Jour. of Genetics, 1938, v. 36, May, pp. 45-51.

A family is described in which one woman is heterozygous both for hemophilia and color blindness, these being derived from different parents. Her only son does not show crossing-over.

T. E. Sanders.

Sevrugina, M. A. **Increase of visual acuity through a conditioned reflex.** Viestnik Opht., 1938, v. 12, pt. 2, p. 266.

The objective of this laboratory investigation was to determine the response of visual acuity to an indifferent stimulus combined with an increase in illumination. The beat of the metronome was used as the indifferent stimulus. The charted results show that this combination of stimuli raises visual acuity 22 percent, and that the increase persists, diminishing gradually, for seven or eight days. Ray K. Daily.

Starkiewicz, Witold, **Afterimages in the periphery of the visual field. The electric theory of the genesis of afterimages.** Klinika Oczna, 1938, v. 16, pt. 3, p. 279.

This exhaustive study reviews in detail the literature on the retinal afterimage and on the electrical retinogram. The author's own contribution to the subject consists in a study of the characteristics of the afterimage in the peripheral field. He finds that in the extreme periphery of the visual field the afterimage persists longest and that its duration is shortest in the middle of the field. Usually the longer the duration of the afterimage the more numerous are its phases. After a description of the photochemical and mechanical retinal changes in the occurrence of this phenomenon he attempts to show a parallelism in the phases of the retinal afterimage and in the phases of the electroretinogram. He believes that this parallelism justifies the hypothesis that the external and internal plexiform layers constitute a condenser for the electric energy generated by the permanent retinal current. The discharge of this energy in the plexiform layers develops an oscillating current, which is a repercussion of the retinal current, and which passes along the visual pathways to the cortex as the afterimage.

Ray K. Daily.

Tassman, I. S. **The use of paredrine in cycloplegia.** Amer. Jour. Ophth., 1938, v. 21, Sept., pp. 1019-1024.

4

OCULAR MOVEMENTS

Agnello, F. **Further contribution to the isolated paralysis of the abducens after fracture of the cranial base.** Riv. Oto-Neuro-Oft., 1937, v. 14, Sept.-Oct., pp. 492-498.

A man of 27 years after a severe trauma of the head remained unconscious for thirty hours and appeared to be affected by left internal strabismus from paralysis of the left external rectus. The author discusses the symptomatology of fractures at the base,

and locates the lesion of the sixth nerve at the apex of the petrous portion of the temporal bone. (Bibliography, 1 figure.) M. Lombardo.

Bocherev, E. M. **Late results of strabismus surgery.** Viestnik Opht., 1938, v. 12, pt. 2, p. 243.

A postoperative follow-up of 233 patients operated upon for strabismus. In 60 percent of the cases the results were satisfactory, 6.4 percent had slight narrowing of the palpebral fissure, one patient had an intractable diplopia, and 24.6 percent had overcorrection or undercorrection. Ray K. Daily.

Borsotti, I. **Considerations on paralysis of the abducens from lumbar puncture.** Riv. Oto-Neuro-Oft., 1938, v. 15, May-June, pp. 205-222.

A girl 2½ years old, affected by meningococcic meningitis, received eleven spinal injections of antimeningococcic serum and six X-ray applications, after which she recovered. After the eighth spinal injection paralysis of the right external rectus was manifest. It disappeared almost entirely after six months. After a lengthy discussion on the present conception of the pathogenetic mechanism of abducens paralysis from lumbar puncture, especially in regard to the intracranial tension, the writer recognizes that paralysis may occur from many factors, one of which is repeated lumbar puncture. (Bibliography, one figure.) M. Lombardo.

Jancke, G. **Differences of strabismus and other conditions in the eyes of an enzygotic pair of twins.** Klin. M.f. Augenh., 1938, v. 101, July, p. 76.

The tests for determination of the enzygotic type of the twins are enumerated in detail. There were concordance of the total pictures of the fundi (although discordance in some points), and discordance of fusion and position of the eyes. It seemed possible that in the same individual convergent and divergent strabismus might alternate.
C. Zimmermann.

Porta, V., and Della Vedova, Jr. **Opticokinetic nystagmus in cerebral affections.** Riv. Oto-Neuro-Oft., 1938, v. 15, May-June, pp. 238-250.

Two girls ten and twelve years of age showed among other symptoms of increased intracranial pressure resulting from subacute hydrocephalus, bilateral disappearance of opticokinetic nystagmus. This, however, reappeared when, after appropriate therapy, the radiologic, ophthalmic, and neurologic syndrome and the associated general symptoms were relieved. (Bibliography, one figure.) M. Lombardo.

Tronconi, V. **Clinical considerations on a case of unilateral sensory and motor ophthalmoplegia.** Riv. Oto-Neuro-Oft., 1938, v. 15, March-April, pp. 168-170.

A woman of fifty years, who had complained of slight frontal headaches, nausea, and vomiting, became affected by pain in the field supplied by the first branch of the trigeminus and then by total ophthalmoplegia on the left side. Of the trigeminus only the nasociliary and frontal branches were affected. From the special distribution of the symptoms it was evident that the origin of the disease resided in a lesion affecting the sensory and motor nerves in their passage through the median portion of the upper orbital fissure or in the circle of Zinn. Regarding the etiology, the writer believes that a lesion of inflammatory type localized in

the portion of the ophthalmic vein within the circle (a periphlebitis), might produce clinical symptomatology such as affected his patient. (Bibliography.)
M. Lombardo.

5

CONJUNCTIVA

Bursuk, G. G. **Methods of conjunctivoplasty.** Viestnik Opht., 1938, v. 12, pt. 2, p. 222.

For restoration of the conjunctival sac the author uses attached flaps from the skin of the lids, in preference to free flaps. (Illustrations.) Ray K. Daily.

Chutko, M. B. **Treatment of ocular phlyctenulosis with blood transfusion.** Viestnik Opht., 1938, v. 12, pt. 1, p. 99.

The author treated phlyctenulosis in children by blood transfusion with satisfactory results. He reviews the literature relative to retinal hemorrhages as complications of blood transfusions, and gives seven brief case histories in which there were such hemorrhages. In six of these the hemorrhage was present prior to the transfusion. Chutko concludes that retinal hemorrhages occur in cases with diseases of the blood or circulatory disturbances, and that the transfusion is not the cause of the retinal hemorrhages, but that where they are present the transfusion may usher in a new attack. Ray K. Daily.

Cornet, Emmanuel. **The obligatory pause in the treatment of certain cases of trachoma.** Rev. Internat. du Trachome, 1938, v. 15, April, p. 84.

There are many occasions where the treatment of trachoma must be interrupted on account of various local and general conditions. If there is an intercurrent conjunctivitis this should be treated while leaving off the usual treatment for trachoma. If there is associated folliculosis the tonsils and adenoids should be removed. Follicular conjunctivitis would contraindicate the more severe treatment. In the event of phlyctenular disease or local syphilis the treatment should be discontinued until these conditions are cured. Antitrachomatous treatment need not be altered in the course of general conditions such as syphilis, tuberculosis, endocrine disturbances, or avitaminosis, but these conditions should be treated concomitantly if the trachoma is to be cured. J. Wesley McKinney.

Delanoë and Sédan, Jean. **Pyrexias and trachoma.** Rev. Internat. du Trachome, 1938, v. 15, Jan., p. 1.

The authors have systematically observed the effect of the hyperpyrexia of such diseases as pneumonia, typhoid, or malaria on trachoma in a large hospital in a locality where the disease is very prevalent. During the course of an illness with high fever the corneal ulcers cleared up rapidly, and pannus regressed and became quiescent to a remarkable degree. They obtained the same result with artificial fever therapy. Hyperpyrexia, however, did not affect the conjunctival manifestations of trachoma. J. Wesley McKinney.

Edison, S. M. **Zinc ionization treatment of trachoma.** Illinois Med. Jour., 1937, v. 71, May, p. 431.

The author reports on his second series of cases, 72 in all. The treatments were given at two-week, three-week, and four-week intervals. The cases included those in the acute and chronic stages and those having corneal ulcers and pannus. All had previously resisted many forms of accepted treatment for a year or longer and zinc ionization resulted in the arrest of the disease after

one or more treatments, excepting in 7 percent of the cases which did not respond. Only edema of the lids and engorgement of the conjunctivas were noticeable after the treatments. Follicles disappeared rapidly, and cicatrices when present became thinner and smoother in most cases. Thickening of the lids and ptosis were noticeably reduced in all cases. Improvement in vision of from 1/10 to 3/10 was noticed in 21 percent of the cases treated. The total percentage discharged as arrested in this and the earlier series was about 41 percent. Twenty-four percent showed amelioration of all subjective symptoms, with notable regression of follicles after only one treatment. The apparatus is inexpensive.

F. M. Crage.

John, I., and Hamburger, F. A. **Results in the attempts to culture the virus of trachoma and paratrachoma.** Graefe's Arch., 1938, v. 138, pt. 6, pp. 760-789.

The discharge and scrapings from the conjunctivas of ten recent cases of paratrachoma and one of recent true trachoma were used. To free this material of contaminating bacteria, filtration was employed. Since this caused too much dilution of the virus, centrifugation of the conjunctival material taken up in Ringer's solution was used. In about fifty percent of the cases the latter succeeded in producing a bacteriologically sterile solution for tissue culture. The allantois of chick embryos was found to be the only culture medium suitable for growth of the virus of trachoma and paratrachoma. Three times, after centrifugation, the inoculated allantois material was applied to the previously scarified conjunctiva of a baboon. This procedure produced intensive symptoms of irritation, lasting only a few days. Microscopic examination of fixed preparations from the tissue cultures or from the baboon's conjunctiva never revealed epithelial cell inclusions or Lindner's initial bodies.

H. D. Lamb.

Kaye, Herbert. **A case of rhinosporidiosis on the eye.** Brit. Jour. Ophth., 1938, v. 22, Aug., pp. 449-455.

This disease being especially rare in the Union of South Africa, a case is reported of a European boy aged sixteen years. Originally the case was reviewed by Simson and Strachan in the South African Journal of Medical Sciences, but the author now follows through from that report. The infection advanced to involve the limbus, whereas when previously reported it had affected only the conjunctiva.

Seen first and treated in February, 1935, it was in October of 1937 that the boy again reported, complaining of a red swelling on the white of his right eye. Examination by slitlamp revealed a recurrence of the original infection. The method of treatment is explained, as is the microscopy of the polyp. The mode of infection, especially in the case noted, is not definitely known, it being suggested by Simson that the violent dust storms of the Transvaal might carry the spore to the eye. Recurrence in the case noted was attributed to the possibility of the sporangia lying dormant in the conjunctival sac until admission to the eye was gained by rubbing. Use of the snare for removal of the polyp is discouraged. The use of gloves during removal is recommended, in order to avoid further infection. (Photomicrographs, references.)

D. F. Harbridge.

Mitzkevich, L. D. **Swimming-pool conjunctivitis.** Viestnik Opht., 1938, v. 16, pt. 3, p. 397.

During a gynecological treatment some secretion from the patient's vagina entered the eye of the gynecologist. The next day the latter appeared with a typical picture of swimming-pool conjunctivitis. The smears were negative for inclusion bodies.

Ray K. Daily.

Rameev, P. S. **Trachoma in combination with spring catarrh.** Viestnik Opht., 1938, v. 12, pt. 2, p. 400.

After seeing ten cases of mixed trachoma and spring catarrh, the author feels that this combination should be kept in mind in areas where both diseases are prevalent. Ray K. Daily.

Rogers, H. R. **Cutaneous and conjunctival diphtheria.** Irish Jour. Med. Science, 1938, 6th series, p. 283.

Eleven cases of cutaneous or conjunctival diphtheria were discovered at the Cork Street Hospital during a period of four months. All but two of the cases entered the hospital for other conditions, such as scarlet fever, measles, and erysipelas. The diphtheritic lesions responded rapidly to antitoxin. There were two deaths from concomitant streptococcal infections.

John C. Long.

Thygeson, Phillips. **A papillary type of Béal's conjunctivitis.** Amer. Jour. Ophth., 1938, v. 21, Sept., pp. 1017-1018.

Trapezontzeva, E. E. **Rickettsia in trachoma.** Viestnik Opht., 1938, v. 12, pt. 2, p. 237.

A report of a laboratory study with inoculations of lice with trachomatous material. The inoculations consisted of a series made directly into lice, a series passed first through the tunica vaginalis of guinea pigs, and a control series of inoculations of nontrachomatous material into lice. The detailed report shows all positive results in the series of direct inoculations. In the other two series there are both positive and negative findings. Ray K. Daily.

6

CORNEA AND SCLERA

Birdwood, G. T. **Keratitis from working with creosote.** Brit. Med. Jour., 1938, July 2, p. 18.

Two gardeners, while creosoting a fence, noticed stinging pain in the eyes. Probably minute drops of creosote entered the eyes although the workmen attributed their discomfort to fumes. Splotchy areas of superficial keratitis occurred which resulted in permanent opacities. John C. Long.

Borsotti, I., and Locatelli, L. **The nerve conditions in experimental leucocytosis by corneal stimuli.** Arch. di Ottal., 1938, v. 45, March-April, p. 55.

The authors have studied the leucocytic curve in rabbits in which they injected melanin into the parenchyma of the cornea. Following the injection there occurred leucocytosis almost double the normal within fifteen to thirty minutes. The increase did not occur after retrobulbar injection of alcohol. Removal of the superior cervical ganglia did not produce an increase in leucocytosis. It is concluded that the stimulus which produced the leucocytosis is carried by sensory pathways.

H. D. Scarney.

Diban, P. A. **Treatment of corneal ulcers with intramuscular injections of 0.25 percent novocaine.** Viestnik Opht., 1938, v. 12, pt. 1, p. 71.

The author modified the Speranski blockade by giving the novocaine injections intramuscularly instead of

around the kidney. Having used this method in 105 cases of corneal ulcer, he shows statistically that the results are superior to those obtained with the usual therapeutic procedures.

Ray K. Daily.

Dimshitz, L. A. **Pathogenesis of some forms of keratitis.** Viestnik Opht., 1938, v. 12, pt. 1, p. 55.

This is a slitlamp study of 66 cases of herpetic keratitis, keratitis disciformis, and keratitis superficialis punctata. The author concludes that these apparently different corneal conditions are very similar morphologically and pathogenetically. Morphologically they have the following features in common: detachment of the corneal epithelium by underlying edema, in the form of fine dots in keratitis punctata, in small vesicles in herpes, and in large vesicles in keratitis disciformis; a pathologic condition of the corneal nerves; and sometimes in herpes corneae and keratitis disciformis a marked edema of the deeper corneal layers, which manifests itself by thickening of the cornea, folds in Descemet's membrane, and clefts in the corneal parenchyma. Pathogenetically the three conditions have in common a pathologic condition of some portion of the nervous system; be it a traumatic involvement of the corneal nerves as in herpetic keratitis and keratitis disciformis, an involvement of the corneal nerves as in herpetic keratitis, or a disturbance of remote nerve functions as in general infections. Clinically they have in common neuralgic pains, disturbances in corneal sensibility, and frequently symmetrical diseased foci. In addition the transition of one form of keratitis into another (such as keratitis dendritica into keratitis disciformis) and the presence of various forms in the same eye indicate a common pathogenesis. The neurotropic element in these conditions is very significant, and explains the occurrence of herpetic keratitis of traumatic origin.

Ray K. Daily.

Garkavi, P. A. **Slitlamp examination of diffuse interstitial keratitis.** Viestnik Opht., 1938, v. 12, pt. 2, p. 160.

A detailed description of the histologic pathology of parenchymatous keratitis and a tabulated report of 37 slitlamp examinations. The conclusions are that the disease is of the corneal stroma, with active participation of the epithelium and endothelium in the process. The process may begin with fine precipitates on the endothelium. The most lasting changes are in the deep layers of the stroma, and in the progressive stage of the disease one frequently sees on the posterior corneal surface precipitates which probably result from endothelial proliferation.

Ray K. Daily.

Krachmalnikov, L. L. **In defense of cauterization in serpiginous corneal ulcers.** Viestnik Opht., 1938, v. 12, pt. 3, p. 404.

The author feels that in the contemporary literature there is a tendency to depreciate the value of cauterization in corneal ulcers. He defends the procedure, because in nine years it has not failed to arrest the progress of the ulcer in one to three days.

Ray K. Daily.

Reiser, K. A. **Histologic studies on the herpetic-inoculation keratitis of rabbits.** Graefe's Arch., 1938, v. 139, pt. 1, pp. 118-164.

The author reports inoculation directly into the rabbit's cornea, from the vesicle contents of herpes zoster eight times, from those of herpes labialis

seven times, scraped-off epithelium of lattice keratitis three times, and from dendritic keratitis ten times. All attempts to inoculate the rabbit's cornea with material from herpes zoster and lattice keratitis were negative. An enucleated eye with lattice keratitis presented folds in Bowman's membrane. From nine cases of dendritic keratitis and six of herpes labialis there developed in the rabbit's cornea, after from one to two days of incubation, branching ulcers, and after from four to five days more, large map-shaped ulcers. Inoculation into the rabbit's brain, was followed by herpetic encephalitis in fourteen out of fifteen cases. The nerves of the corneal parenchyma showed no characteristic pathologic change in herpetic-inoculation keratitis.

H. D. Lamb.

Shapiro, E. A. **The effect of Speranski's blocking on corneal sensitivity.** Viestnik Opht., 1938, v. 12, pt. 1, p. 67.

Having used Frey's hair for testing corneal sensitivity, the author shows through tabulated case reports that novocaine blocking administered in cases of keratitis with reduced corneal sensitivity definitely raises the sensitivity. Ray K. Daily.

Sie-Boen-Lian. **Bitot's spot as not a part of xerophthalmia.** Graefe's Arch., 1938, v. 139, pt. 1, pp. 17-21.

In two years, the author has observed nineteen cases (eighteen males) of Bitot's spot in which deficiency of vitamin A as the etiologic factor could be excluded. One patient was 35 years old and another 44 years, but the age as a rule was from 11 to 30 years. The majority of the patients stated that the condition was present in childhood. The fleck might be unpigmented or pigmented and was usually large or moderately large (2 to 15 mm. in diameter). H. D. Lamb.

Zazibin, H., and Plitas, P. C. **Corneal repair.** Viestnik Opht., 1938, v. 12, pt. 2, p. 181.

A laboratory study on animals. Sections of the cornea were excised to but not through Descemet's membrane, and the entire corneas were examined histologically during the various periods of corneal repair. The author emphasizes the fact that the appearance of large numbers of connective-tissue cells in the wound is due to two processes: the change of old fixed cells into young cells capable of motion, and formation of new fixed cells by direct or indirect proliferation of fibroblasts and fibrocytes. The author also records the sending out of fibers from fixed cells in the process of differentiation from fibroblasts; these fibers when cemented together forming the layers of corneal stroma. Ray K. Daily.

7

UVEAL TRACT, SYMPATHETIC DISEASE, AND AQUEOUS HUMOR

Kaminskaja, Z. A. **Peripheral innervation of the iris.** Viestnik Opht., 1938, v. 16, pt. 1, p. 35.

The response of the isolated iris to adrenalin justifies the conclusion that the iris possesses a peripheral innervation independent of central nervous control. The object of this study was to narcotize this peripheral innervation and to determine the subsequent reaction of the iris to adrenalin. Nicotine injected subconjunctivally or injected into the anterior chamber produces a miosis which is not at all or only slightly affected by adrenalin. Soon afterward there develops a dystrophy in a sector-shaped area of the iris ad-

jacent to the point of injection, manifested by an irregular distribution of pigment in its stroma. To the author this phenomenon indicates that the peripheral innervation of the iris plays an important part in the metabolism of the eye. Ray K. Daily.

Lacarrère, J. L. **First trial of retroiridectomy.** Trabalhos do Primeiro Cong. Brasileiro de Ophth., 1936, v. 1, pp. 403-405.

On a blind eye with iris bombé the author wished to perform an operation with the sole purpose of preserving the eye by permanent reduction of ocular tension. Instead of entering the anterior chamber in front of the iris, which was practically impossible, he entered behind the iris and performed a large iridectomy through this incision. The iris was found to be atrophic, and adherent both to the cornea at the trabeculum and to the capsule of a cataractous lens. The postoperative course was uneventful, and the eye regained and kept normal tension.

Ramon Castroviejo.

Mass, M. M., and Shepkalova, V. M. **Sarcoma and sympathetic ophthalmia.** Viestnik Opht., 1938, v. 16, pt. 2, p. 390. (See Section 15, Tumors.)

Orlov, K. X. **Gonococcemia and the eye.** Viestnik Opht., 1938, v. 12, pt. 1, p. 22.

The author believes that gonorrheal iritis is caused by a metastasis of the organism and not by its toxins, and he urges the use of the complement fixation reaction in diagnosis. Five case reports illustrate the therapeutic effectiveness of sodium salicylate and collargol administered intravenously.

Ray K. Daily.

Pesce, Girolamo. **Contribution to the study of paradoxic pupillary reaction to light.** Riv. Oto-Neuro-Oft., 1938, v. 15, May-June, pp. 223-237.

A man seventy years of age became suddenly blind in the right eye from thrombosis of the central vein. At the examination it was noted that on testing the pupillary reaction to light the pupil became dilated. The pupillary diameter which at rest was 3.8 mm. reached a size of 6.9 to 7.5 mm. Miosis was present in O.D. on illuminating O.S., and slight miosis in O.S. when O.D. was illuminated. The condition lasted for about a week. Lues was excluded as a factor and the phenomenon was attributed to mesencephalic softening from thrombotic foci. (Bibliography.) M. Lombardo.

9

CRYSTALLINE LENS

Cavallacci, G. **Some cases of endocrine cataract. Clinical and biomicroscopic contribution.** Arch. di Ottal., 1938, v. 45, May-June, p. 133.

The author describes three cases of endocrine cataract, two in subjects with clinical evidence of hypophyseal dysfunction and one in a subject of typical ovarian insufficiency. In all three cases laboratory examinations produced evidence of abnormal variations in calcium and potassium. The lenticular change is considered by the author to be due to an alteration in the fluid equilibrium of the eye. The biomicroscopic findings also confirm the characteristic morphologic findings of endocrine cataract. (8 plates.)

H. D. Scarney.

Jackson, Edward. **Applied mechanics of cataract extraction.** Amer. Jour. Ophth., 1938, v. 21, Sept., pp. 1011-1016; also Trans. Western Ophth. Soc., 1937, 4th mtg.

Paez Allende, Francisco. **Intracapsular extraction of cataract by means of the Snellen loop.** Arch. de Oft. de Buenos Aires, 1938, v. 13, Feb., p. 89.

After citing the well known advantages of intracapsular extraction and the objections to the usual methods of this operation, the author describes in detail his maneuver of extracting the lens in its capsule. Employing a retrobulbar injection of novocaine and performing an external canthotomy he proceeds in orthodox fashion up to the point of expression of the lens. The lens is then dislocated by making pressure with a strabismus hook and is delivered with the Snellen loop. Conjunctival sutures of fine silk are used. The visual results of fifteen cases are appended. Edward P. Burch.

Urbanek, J., and Albrecht, W. **Vitamin C and age.** Zeit. f. Augenh., 1938, v. 95, June, p. 129.

The author fed and injected vitamin C to a point of saturation in 150 patients daily for several weeks. Patients can take 100 to 300 mg. of ascorbic acid per day before the urinary output—normally 6 to 7 mgs. per day—increases at all. Titrimetric measurement of the urinary output is a simple procedure. After three days of 300-mg. intake, a normal individual usually excreted 80 to 90 percent of the ingested amount. It was found that cataract patients from an old peoples' home were poorer in vitamin C than private patients. The senile organism was poorer than the juvenile. Children excreted vitamin C sooner than the aged. The authors find that when the organism is saturated with ascorbic acid the eye operated on for cataract is much less apt to have postoperative hemorrhage.

F. Herbert Haessler.

10

RETINA AND VITREOUS

Ascher, Karl. **The measurement of the pressure in the intraocular blood vessels.** Graefe's Arch., 1938, v. 139, pt. 1, pp. 62-79.

Using the method of Bailliart, the author found no essential change in the diastolic pressure of the retinal arteries when the patient, in an air chamber, was submitted to increase or diminution of atmospheric pressure. Increase of intraocular tension and increase of the general blood pressure caused increase of the diastolic tension in the retinal arteries. Out of 85 cases of suspected increased intracranial pressure, 65 presented a high figure for the diastolic tension in the retinal arteries. The diastolic tension in the central retinal artery showed no essential difference in 23 cases of tabes dorsalis with optic atrophy, as compared with 28 cases of tabes dorsalis without optic atrophy. H. D. Lamb.

Bartels, M. **The cause of retinal detachment, with particular regard to the referee in compensable injuries.** Zeit. f. Augenh., 1938, v. 95, July, p. 177.

Bartels describes the manifestations which, after experience with many hundred cases, he has come to consider essential for an opinion on the cause of a retinal detachment. He emphasizes the fact that a tear may exist for a long time before detachment follows and that a flat or small detachment immediately after trauma may have escaped detection on first examination.

In aphakic eyes detachment occurs more readily, but Bartels denies that every aphakic myopic eye will sooner or later suffer retinal detachment. It is very important to study thoroughly the

fellow eye for signs of degenerative change to help determine whether a detachment is more likely to be spontaneous or is ascribable to trauma. Heredity plays no part. Choroiditis is apt to prevent, rather than cause, retinal detachment. In unquestionably sound eyes the author has seen detachment after penetrating as well as blunt trauma. Concussion without direct trauma to the eyeball is a doubtful cause of detachment.

F. Herbert Haessler.

Bietti, Giambattista. **Researches on the action of some drugs on the pressure of the central artery of the retina, with special regard to adrenalin.** Riv. Oto-Neuro-Oft., 1938, v. 15, March-April, pp. 113-160.

The experiments were made in persons of 13 to 71 years, using adrenalin, sympathol, lymphoganglin, ergotamine, and caffeine. The retinal pressure (Bailliart dynamometer), the general blood pressure, and the ocular tension were tested from time to time after use of each drug either hypodermically or endovenously. The results are given in tabulated form. The changes in retinal arterial pressure were not always in harmony with the changes that a drug produced in the general blood circle. The retinal arterial pressure frequently followed the lowering but not so frequently the elevation of general blood pressure, which result the authors are disposed to attribute to the fact that the central retinal artery is less responsive to the drugs. The central artery of the retina, perhaps because easily explorable and because it can determine pressure changes with precision, lends itself to furnishing an index of the behavior of the pressure of the cerebral blood vessels, which react similarly to the retinal ones in various clinical and experimental conditions. (Bibliography.) M. Lombardo.

Blumenthal, C. J. **Withdrawal of interretinal fluid in detachment operation by simple suction apparatus.** Brit. Jour. Ophth., 1938, v. 22, Sept., pp. 542-543.

A simple suction apparatus is described which reduces the element of danger in withdrawal of interretinal fluid. Removal of the fluid at operation, rather than dependence on spontaneous leakage or absorption, is recommended as indicated by a view of the fundus on the day after the operation. Withdrawal of the fluid at the time of operation also avoids the "floating up" of the area of coagulation due to unavoidable change in the position of the patient's head in the 48-hour postoperative period. (Illustrated.) D. F. Harbridge.

Buking-Gerung, Z. A. **Rare anomalies of the fundus.** Viestnik Opht., 1938, v. 12, pt. 2, p. 234.

A report of two cases. In one the fundus showed a structure resembling a cyst, which the author after study concludes was a retinal cyst formed at the time of closure of the embryonic optic cup. The second case is an unusual form of persistent remains of Cloquet's canal and the hyaloid artery.

Ray K. Daily.

Ivanova, E. M. **Pericarotid sympathectomy in retinitis pigmentosa.** Viestnik Opht., 1938, v. 12, pt. 3, p. 311.

A review of the literature and an analysis of twenty cases, ten of which had bilateral and ten unilateral pericarotid sympathectomies, relative to functional and objective results. Of the unilaterally operated cases vision remained unchanged in six and improved in four. Of the bilaterally operated cases vision remained unchanged in five

and improved in five. Histologic study of the removed pericarotid nerve fibers, correlated with the clinical results, demonstrated that in the cases in which the nerve fibers were normal the operation was beneficial. Where the nerves were degenerated the effect of the operation was negative. In cases with initial signs of degeneration in the nerves, the improvement in central vision following the operation was not permanent, but vision remained better than prior to the operation. (Illustrations.) Ray K. Daily.

Nathan, David. **Maculo-cerebral degeneration.** Amer. Jour. Ophth., 1938, v. 21, Sept., pp. 1029-1031.

Odintzov, V. P., and Shelagurov, A. A. **Etiology of retinitis pigmentosa.** Viestnik Opht., 1938, v. 12, pt. 1, p. 3.

Six patients with retinitis pigmentosa were subjected to a very exhaustive laboratory study relative to liver function; in none of these did the tests show deviations from normal. After reviewing the literature on the anatomic pathology of retinitis pigmentosa, Odentzov offers the following conception of its etiology: A hereditary defective anlage of the primary optic vesicle and its blood vessels leads to development of unstable retinal nerve elements. They function normally for a time, and then undergo degeneration because of insufficient nutrition. The severity of the disease depends on the degree of involvement of the various elements. In severe involvement of the retinal elements, rapid development of the disease may take place in early youth even without much vascular disturbance. In mild involvement of the retinal elements visual disturbances may not appear until after middle age. Therapy of the disease must be directed toward improving retinal nutrition through vasodilatation. No therapeutic or surgical procedures can cure the disease, but they can delay the process, and even that is of great benefit to the patient. Ray K. Daily.

Wilczek, M. **A case of unusual vitreous strings.** Graefe's Arch., 1938, v. 139, pt. 1, pp. 22-31.

A boy fifteen years old presented in the right eye, by ophthalmoscopic examination, a rather thick grayish cord extending from the inferior half of the optic disc, far anteriorly into the axis of the vitreous. At its anterior extremity there were present two vesicular-like dilatations. From the latter, spread anteriorly numerous fine filaments. In addition, similar grayish cords with vesicular-like endings and a spread of fine filaments extended into the vitreous from three densely pigmented foci in the fundus. Three partly pigmented foci lay at the macula and in line inferior to it. These foci were irregularly connected by delicate grayish-white bands, lying close but anterior to the retinal blood vessels and presenting the markings of atypical colobomas. The author thinks this tissue on the retina must be considered as persistent embryonal tissue of the fetal fissure. The strings inserted into the retina are probably of glial origin. H. D. Lamb.

11

OPTIC NERVE AND TOXIC AMBLYOPIAS

Bietti, Giambattista. **Considerations on the appearance of papilledema in myopic eyes.** Riv. Oto-Neuro-Oft., 1938, v. 15, Jan.-Feb., pp. 47-58.

A woman of 38 years who accepted +2.00 D. sphere for O.D. and −15.00 D. sphere for O.S. became affected with

symptoms of intracranial tumor; including marked edema of the right papilla and a ring of edema around the left papilla. During the further seven weeks until the time of her death the edema reached a high level in the hyperopic eye and always appeared to be very slight in the myopic eye. A detailed discussion is given of the anatomic conditions of the optic nerve and papilla and their relation with the scleral canal in myopic eyes, as inhibiting full development of edema of the disc in such cases. (3 figures.)

M. Lombardo.

Boschi, G. **The diacerebrospinal treatment of neuritis and atrophy of the optic nerve.** Riv. Oto-Neuro-Oft., 1938, v. 15, March-April, pp. 178-181.

In a preliminary note the writer suggests provoking a meningeal reaction or an aseptic meningitis to activate neuromeningeal drainage. The proposal is based on his conception of the mechanism of production and circulation of the cerebrospinal fluid. The technique varies from simple lumbar puncture to the intraspinal injection of different substances among which are air and the cerebrospinal fluid of the patient. Visual improvement is reported in cases of disseminated sclerosis, tabetic atrophy, and retrobulbar neuritis.

M. Lombardo.

Chiatellino, A., and Prister, B. **Experimental researches on the pathogenesis of papillary stasis.** Riv. Oto-Neuro-Oft., 1938, v. 15, March-April, pp. 108-112.

To determine the source of the formation of edema of the papilla other than from a mechanical cause, the authors made various experiments in dogs. They ligated the optic nerve in the orbit or intracranially, or provoked compression of the nerve in the optic canal, or stimulated the nerve with various substances; or injected some substance into the epidural space in order to provoke a rapid increase of intracranial pressure. Some of the experiments gave an ophthalmic picture similar to papilledema. The authors think, however, that these experiments do not justify definite conclusions. The experiments are to be continued.

M. Lombardo.

Clivio, Cesare. **An operated case of arachnoiditis with atrophy of the optic nerves.** Riv. Oto-Neuro-Oft., 1938, v. 15, March-April, pp. 104-107.

A boy of nine years affected by progressive loss of vision, headaches, and vomiting, and totally blind at the time of examination, showed complete atrophy of the optic nerves and on radiography no sign of increased intracranial pressure but signs of calcification above the sella turcica. At operation the optic nerves, the chiasm, and adjacent structures were found surrounded by adhesions which were removed. A slight improvement of vision followed. Early diagnosis and proper operative treatment may enable this class of patient to retain vision. M. Lombardo.

Dolganov, V. H. **Quinine poisoning.** Viestnik Opht., 1938, v. 12, pt. 1, p. 46.

Three case reports illustrate the divergence between ophthalmoscopic and functional findings in cases of quinine poisoning. Dolganov explains the fairly good visual acuity with atrophic discs by the ability of the choroidal system to assume the function of the spastic retinal vessels. The spasm of the retinal vessels produces first a retinitis, and then a neuritis followed by secondary optic atrophy. Therapy should be directed toward improving the vascular

nutrition of the retina, and should be continued for a long period in spite of initial negative results.

Ray K. Daily.

Glees, M. **Changes of the optic nerves from arteriosclerosis similar to the syndrome of Foster Kennedy.** Klin. M. f. Augenh., 1938, v. 100, June, p. 865.

The chief symptoms of the Foster Kennedy syndrome are choked disc in one eye and simple atrophy of the optic nerve with central scotoma in the other. Three cases of this type, but without central scotoma, are described. They were caused by arteriosclerosis, not by a new formation. The process is ascribed to pressure of the arteriosclerotic carotids on the optic nerves, with local disturbances of circulation and nutrition. In all cases the nasal or binasal halves of the visual fields were affected. Only in one case were the carotids visible in the roentgen picture. C. Zimmermann.

Junqueira, G., and Silveira, A. **Tumor of the pontocerebral angle with late appearance of papilledema.** Trabalhos do Primeiro Cong. Brasileiro de Ophth., 1936, v. 1, pp. 367-384.

The papilledema did not manifest itself until very late in the course of the growth of the tumor, in spite of the increased intracranial tension that was present for some time previous to the appearance of the papilledema. (32 references.) Ramon Castroviejo.

Leinfelder, P. J. **Pathologic changes in amblyopia following tryparsamide therapy.** Jour. Amer. Med. Assoc., 1938, v. 111, Oct. 1, p. 1276; also Trans. Sec. on Ophth., Amer. Med. Assoc., 1938, 89th mtg.

A man aged 52 years became blind two days after an injection of one gram of tryparsamide and died nine days later. Acute degeneration of the retinal ganglion cells and of the cells in the innermost portion of the inner nuclear layer was found. There was no evidence of acute primary degeneration of the optic nerve or tracts. General pathologic changes due to arsenic changes were not observed. (4 figures, discussion.) George H. Stine.

12

VISUAL TRACTS AND CENTERS

Brunetti, F., and Spangaro, C. **Histogenetic interpretation of hypophyseal duct tumors.** Riv. Oto-Neuro-Oft., 1937, v. 14, Sept.-Oct., pp. 435-483.

A man of 51 years had become blind in the right eye at the age of 27 years, after attacks of headache in the frontoorbital region. Lately visual disturbances had started in the left eye, preceded by intense headaches and vomiting. The patient had bilateral strabismus when the eyes were at rest, with left vision of 6/10 and absence of the temporal visual field, except as to the macular region. Radiography showed signs of hypertension in the anterior cranial fossa, and enlargement of the sella. At operation a mass was found occupying the sellar cavity. The writers give a lengthy discussion on the histology and histogenesis of the neoplasm. It was derived from the hypophysis and was a squamous epithelioma having a special epidermoid character, as shown by corneal desquamation, the presence of keratin, melanotic pigment, and cholesterin. This type of hypophysis tumor includes two groups: one without epidermoid characteristics (Erdheim's tumor) and the other showing epidermoid characteristics. Both types have a common origin, that is, they are blastomas de-

rived from non-differentiated inclusions of the pharyngeal-hypophyseal duct. (Bibliography, 5 figures.)

M. Lombardo.

Evans, J. N., and McFarland, R. A. **The effects of oxygen deprivation on the central visual field.** Amer. Jour. Ophth., 1938, v. 21, Sept., pp. 968-980.

François, J. **Syphilitic optico-chiasmic arachnoiditis and pyretotherapy.** Bull. Soc. Franç. d'Opht., 1937, v. 50, p. 185.

A 29-year-old patient with acquired syphilis had received regular treatment for a year and a half. Under observation there was rapid loss of vision in the right eye to zero. There was right papilledema that measured 3 D. The left vision was 10/10, and the disc was in an early state of edema and swelling. There was left central scotoma for colors. The general and neurologic examinations and blood and spinal-fluid Wassermann tests were normal. In spite of bismuth and arsenic injections, and cyanide of mercury intravenously, the vision in the left eye became worse and dropped to zero. Vision did not improve after a cranial decompression operation, and an exploratory operation was done upon the optic nerves and chiasm. Adhesions and increased fluid were found about the left optic nerve which was swollen to twice its normal size. The sheath was incised. The left vision improved to counting of fingers at 4 meters and then diminished. Sulphur in oil was given, to cause a rise in temperature. The left vision improved to 2/3 and so remained. The nerve head appeared atrophic. The peripheral visual field remained full. There was no recovery of vision in the right eye. The nerve was atrophic.

Clarence W. Rainey.

Grandi, G. **Chiasmatic syndrome from craniopharyngioma.** Riv. Oto-Neuro-Oft., 1938, v. 15, Jan.-Feb., pp. 72-78.

A woman of 23 years showed: O.D., papilla pale in its temporal half, temporal hemiachromatopsia, V. 15/10; O.S., papilla pale throughout but more so in its temporal half, venous blood vessels distended, large central scotoma of the temporal-hemianopsic type, temporal hemiachromatopsia. Radiography showed an increase of the antero-posterior diameter. At operation by the transfrontal endocranial route, a mass was seen arising from the sella. It was removed by curettement and diathermocoagulation. Examination a month later showed recovery as to all symptoms in O.D., no improvement in O.S. (Bibliography, 3 figures.)

M. Lombardo.

Paleari, Antonio. **Osteochondroma of the cranial base with diencephalic syndrome.** Riv. Oto-Neuro-Oft., 1938, v. 15, Jan.-Feb., pp. 59-71.

As a first sign of intracranial neoplasm of the base in the left parasellar region, a girl of fifteen years had suffered since the age of three years from diplopia which was due to paralysis of the left abducens. This was followed by progressive exophthalmos of the left eye, a right-sided motor hemisyndrome, and an increase in weight. The margins of both optic discs were elevated. The outlines of the sella were not visible on the radiography, but signs of calcification were present in the left parasellar region. At autopsy a neoplastic mass was found at the base of the left hemisphere, in relation with the left median fossa. The growth was adherent to the bone. (Bibliography, 6 figures.)

M. Lombardo.

Rubino, A. **Chiasmatic syndrome from craniopharyngioma in a luetic patient.** Riv. Oto-Neuro-Oft., 1937, v. 14, Sept.-Oct., pp. 484-491.

A woman of 39 years, a luetic, had become totally blind in the course of a few days a year previously. After antiluetic treatment some vision came back, and at the time of the last examination it had been right 7/10 and left 5/10, with complete bitemporal hemianopsia and signs of simple optic atrophy. Radiography showed enlargement of the sella. At operation an intrasellar mass was found, which was diagnosed as a cystic craniopharyngioma. Improvement of central vision and of the visual fields followed the operation. The writer emphasizes the importance of early surgery, even as a simple exploratory measure in doubtful cases, since benefit may be obtained without exposing the patient to great operative risk. (Bibliography, 3 figures.)

M. Lombardo.

Spotnitz, H., and Elsberg, C. A. **The relative refractory period of olfaction and of vision.** Bull. Neurolog. Inst. New York, 1938, v. 7, March, p. 78.

The relative refractory period is the time following exposure to a powerful stimulus during which a weak stimulus is not recognized. This relative refractory period in the eye corresponds to the time required for dark adaptation. The duration of this period is, in all probability, primarily determined by the centrally situated neurons; and is frequently prolonged in the presence of supratentorial intracerebral growths, the duration likely being a measure of the number and degree of degeneration of the neurons involved.

John C. Long.

13

EYEBALL AND ORBIT

Caramazza, F. **Phlegmon of the orbit.** Riv. Oto-Neuro-Oft., 1938, v. 15, Jan.-Feb., pp. 1-46.

Twelve cases of lesion of the orbit are reported. A serous tenonitis or cellulitis recovered spontaneously. There were two cases of lid fistula one from sinusitis and one from mucocele of the frontal sinus. There were further nine cases of phlegmon of the orbit. Five of these were in male patients whose ages varied from five months to 48 years, four in females varying from eleven to 65 years. Five of the phlegmons originated from the sinuses: two from the antrum, two from the ethmoid and maxillary sinuses, and one from the frontal ethmoid and maxillary sinuses. Of the other phlegmons one originated from suppurative metastatic choroiditis, one manifested itself in the presence of a hordeolum, and two were of unknown origin. The mortality among the phlegmon cases was high, reaching 44.5 percent. The writer discusses the treatment of the encysted and diffuse types. In the latter the treatments varied from simple opening of the orbit to exenteration combined with opening and drainage of the periorbital sinuses. (Bibliography, 27 figures.) M. Lombardo.

Lewis, P. M. **Inflammatory pseudotumor of the orbit.** Amer. Jour Ophth., 1938, v. 21, Sept., pp. 991-995; also Trans. Amer. Ophth. Soc., 1937, v. 35, p. 328.

Loepp, W. **Contributions to roentgen diagnosis in ophthalmology.** Zeit. f. Augenh., 1938, v. 95, July, p. 188.

In this essay by a roentgenologist, case histories of six patients are briefly reported in which etiologic diagnosis of

an exophthalmos was made by roentgenologic study. The cases, chosen not for any unusual manifestations but as rather typical illustrative examples, were as follows: mucocele of the sphenoid sinus, intraorbital lipoid granloma, neurofibromatosis of Recklinghausen, epipharyngeal tumor, scurvy, and purulent inflammation in the ear with negative otologic examination.

F. Herbert Haessler.

14

EYELIDS AND LACRIMAL APPARATUS

Esser, J. F. S. **Biological frontal flaps in ophthalmology.** Amer. Jour. Ophth., 1938, v. 21, Sept., pp. 963-967.

Lijo Pavia, J. **Marked bilateral inflammatory ectropion.** Rev. Oto-Neuro-Oft., 1938, v. 13, March, p. 83.

The author reports a case of marked ectropion in a two-year-old child. When first observed the patient was suffering from keratoconjunctivitis of tuberculous origin. A purulent conjunctivitis developed from which staphylocci were recovered. This condition subsided under antiseptic measures, but reinfection with gonococcus organism occurred. Three weeks after onset of this infection the ectropion became manifest, and impetigo of the cheeks set in. Vaccine therapy was instituted against both the Neisserian and the pyogenic infections, and the usual local measures were employed. The exposed conjunctiva was protected by a paraffin shield. The author proposes that this type of ectropion be designated as inflammatory in contradistinction to the four classical types. Edward P. Burch.

Saint-Martin, R. de. **A modification of Motais's ptosis operation.** Ann.d'-Ocul., 1938, v. 175, Aug., pp. 589-596.

The author believes that an operation of the Motais type is the best for ptosis. In order to avoid the notching which so often occurs at the center of the upper lid, he proposes attaching the superior-rectus slip at either side of the center of the lid. John M. McLean.

Tita, C. **Concerning a case of oriental button of the palpebrum. Inoculation of the leishmania tissue in the eyes of dogs.** Arch. di Ottal., 1938, v. 45, March-April, p. 86.

A case is reported positive for leishmania. Positive cultures have been obtained by transplanting the organism from human tissue to dogs, but in the present case no positive findings were obtained following inoculation into the eye tissues of dogs. H. D. Scarney.

15

TUMORS

Damel, C. S., and Villegas, R. R. **Cylindroma of the orbit,** Arch. de Oft. de Buenos Aires, 1938, v. 13, Feb., p. 62.

A case of orbital tumor occurring in a man of 58 years is reported. Histopathologic studies revealed a cylindroma with its origin in the lacrimal gland. The clinical history, physical, laboratory, and eye examinations are given in detail as well as a description of the operative approach, which consisted of a Golovine incision afterward modified into a Krönlein because of difficulties of exposure. Edward P. Burch.

Frost, A. D. **Chloroma.** Amer. Jour. Ophth., 1938, v. 21, Sept., pp. 997-1008; also Trans. Amer. Acad. Ophth. and Otolaryng., 1937, 42nd mtg., p. 123.

Kreibig, W. **Rare orbital tumors.** Zeit. f. Augenh., 1938, v. 95, June, p. 113.

The author describes two rare orbital

tumors clinically and histologically. The first occurred in a woman of 26 years who had had unilateral exophthalmos since birth. In time this had increased. Because of pain, a biopsy was done in 1930 and only fatty tissue was obtained. In 1937 excision was undertaken because of increased pain. The globe was displaced 1 cm. forward and down over the inferior orbital margin. At operation under local anesthesia after incision under the eyebrow and through the fascia, an encapsulated mass was easily dissected free and was found to be attached firmly to a 2-mm. round strand at its two poles. Following this strand into the orbit, two more tumor masses were found on it and excised with it. Four months later the upper lid was freely movable. There was slight endophthalmos and complete anesthesia of the right forehead. The astigmatism formerly present has disappeared. It is clear that the tumors were on the right supraorbital nerve, which was excised at operation. Histologic preparations revealed epineural and perineural connective-tissue proliferation. The nerve itself was normal. Neural neoplasms are rare in the lids and orbit. The most common are plexiform neuromas which may be the only manifestation of a neurofibromatosis of Recklinghausen or may occur as part of a generalized neurofibromatosis.

The other tumor was a cholesteatoma of the superior orbital margin in a man of 55 years. The freely movable globe was displaced forward, down, and out. The lid was freely movable. Two thirds of the orbital margin was occupied by a hard nodular mass. X ray revealed destruction of the bone without involvement of the frontal sinus. Treatment by mercurial inunctions for four months was of no avail. Operation was urged by the roentgenologist, who made a diagnosis of cholesteatoma and feared perforation of the skull. The friable masses removed surgically consisted of cholesterin and granulation tissue. The most important histologic characteristic, squamous epithelium, could not be identified. A diagnosis of destructive lipogranuloma was made. Cholesteatoma may occur anywhere on the cranial vault but is particularly frequent on lateral orbital margin and roof. F. Herbert Haessler.

Mass, M. M., and Shepkalova, V. M. **Sarcoma and sympathetic ophthalmia.** Viestnik Opht., 1938, v. 16, pt. 2, p. 390.

A man 58 years old developed sympathetic ophthalmia of the right eye as complication of a sarcoma in the left eye. The author urges that in cases of atrophy of an eyeball following spontaneous iridocyclitis with beginning iridocyclitis in the other eye, suggestive of sympathetic ophthalmia, one should consider the possibility of a necrotic sarcoma in the primarily involved eye, and insist on enucleation for diagnostic purposes.

Ray K. Daily.

Michaïl, D. **Symmetrical lymphomata of the lacrimal glands.** Ann. d'Ocul., 1938, v. 175, Aug., pp. 565-581.

Symmetrical swelling of the lacrimal glands, which when accompanied by swelling of the salivary glands is usually classed as Mikulicz' disease, is sufficiently rare to be poorly understood. Some authors consider the affection tuberculosis; some, syphilis; but the majority believe it to be a lymphoid hyperplasia of undetermined nature.

Two cases are reported. One patient developed swelling of both lacrimal glands after typhoid fever. Biopsies showed nonspecific lymphoid hyper-

plasia but suggested neither tuberculosis nor syphilis. The other patient slowly developed tumor masses in both lacrimal glands. Both tumors were removed but in two months the tumor recurred in one orbit, together with bilateral infiltrating lesions of sclera, cornea, and conjunctiva, nodular lesions of nasopharynx and palate, swelling of the salivary glands, and nodular lesions in the skin. Biopsies demonstrated lymphoid infiltration of the lacrimal gland and similar cellular nodules around the hair follicles and the sweat and sebaceous glands of the skin. This case suggests generalized sarcomatosis or an aleukemic leukemia, for the blood picture was normal.

John M. McLean.

Tristaino, L., and Keusch, I. **Glioma of the optic nerve.** Riv. Oto-Neuro-Oft., 1938, v. 15, May-June, pp. 251-264.

A boy of two years, who at the age of seven months had had his right eye enucleated for retrobulbar or intracanalicular tumor, and had had the orbit exenterated twice for relapse, died after the last operation from streptococcus meningitis. The post-mortem examination revealed that the right optic nerve had been destroyed, and a neoformation was found on the lower aspect of the frontal lobes. Taking into account the course of the disease including exophthalmos, unilateral papilledema, radiographic findings, the findings at autopsy, the histopathologic findings, and the age of the patient, the author concludes that the case was one of blastoma of the optic nerve, of the gliomatous type, which had undergone diffusion or metastasis to the frontal lobe and had been complicated by purulent meningitis secondary to a suppurative process in the orbit. (19 figures.) M. Lombardo.

NEWS ITEMS

Edited by Dr. H. Rommel Hildreth
640 S. Kingshighway, Saint Louis

News items should reach the Editor by the twelfth of the month

Miscellaneous

On October 11, 1938, a group of ophthalmologists met in Washington, D.C., and organized the American Orthoptic Council. Three members had been appointed for this purpose by each of the three national ophthalmological societies as follows: for the American Ophthalmological Society, Dr. John Dunnington, Dr. Alfred Cowan, Dr. Edwin Dunphy; for the Section of Ophthalmology of the A.M.A., Dr. Derrick Vail, Dr. Everett Goar, Dr. Harold Gifford; for the American Academy of Ophthalmology and Oto-laryngology, Dr. LeGrand Hardy, Dr. Grady Clay, Dr. Avery Prangen. The following officers were elected: president, Dr. LeGrand Hardy, vice-president, Dr. Derrick Vail, secretary-treasurer, Dr. Edwin Dunphy.

It was voted that this council be known as the American Orthoptic Council and that its function shall be the regulation of the principles and practice of orthoptic training. The following resolutions were adopted: 1. Orthoptics has a definite place in ophthalmology. 2. Ophthalmologists should know more about this subject. 3. Ophthalmologists, in general, are unable to carry out this work themselves, and therefore technicians are needed. 4. The national societies through the Orthoptic Council should take over the training of technicians, and a central station should be established for such training.

By-laws were adopted which provide that the council shall be composed of nine executive members, three from each national society, and three associate members, who shall be orthoptic technicians or instructors, to be selected by the nine executive members. It was also provided that examination shall be held at least once a year, and that a certificate of proficiency shall be awarded to those candidates who have satisfied the board of examiners. A set of rules was also adopted governing the training and practice of orthoptic technicians, and a code of ethics governing deportment of such technicians. A syllabus for student orthoptic technicians will be issued shortly.

The George Washington University School of Medicine, Washington, D.C., announces an intensive postgraduate course in ophthalmology, April 10 to 15, 1939, inclusive. In addition there is offered a special practical course limited to 25 participants, April 4 to 8, 1939, inclusive. These courses are given by the resident staffs of George Washington University School of Medicine and the Army Medical Museum, and by 17 guest lecturers. For further details apply to the Secretary, Miss Louisa G. Wells, 927 17th Street N.W., Washington, D.C.

Wayne University College of Medicine, Detroit, Michigan, announces a postgraduate seminar in biomicroscopy for licensed physicians who are specializing in diseases of the eye, December 12th to 17th, inclusive, under the direction of Dr. Parker Heath, professor of ophthalmology. For further information and to make application, address the Dean, Wayne University College of Medicine, 1512 St. Antoine Street, Detroit, Michigan.

Beginning with the issue for August, 1938, Graefe's Archiv für Ophthalmologie and the Archiv für Augenheilkunde were combined under the name of the former journal.

Societies

The Section on Ophthalmology of the American Medical Association has received an invitation from the Ophthalmological Society of Australia to send a representative to its first annual general meeting to be held in Melbourne, Australia, from April 3 to 6, 1939. If any Fellow of the American Medical Association, Section on Ophthalmology, is planning a world trip in 1939 the Section would be interested to hear from him so that he could be appointed our representative. Derrick Vail, Secretary.

The Section on Ophthalmology of the Philadelphia College of Physicians has established the deSchweinitz Lecture, which Dr. Edward Jackson gave on November 17th.

The Philadelphia County Medical Society Eye Section met on November 3, 1938. Dr. Edward S. Gifford presented "Psychological development of ocular neurosis." Dr. James Watson White, New York City, presented "Clinical problems of ocular motor anomalies."

Personals

The present address of Dr. Laura A. Lane is requested by the manuscript editor (Miss Emma S. Buss, 6820 Delmar Boulevard, Saint Louis). Any one knowing Dr. Lane's address is asked to send it in.

JAN 5 1939

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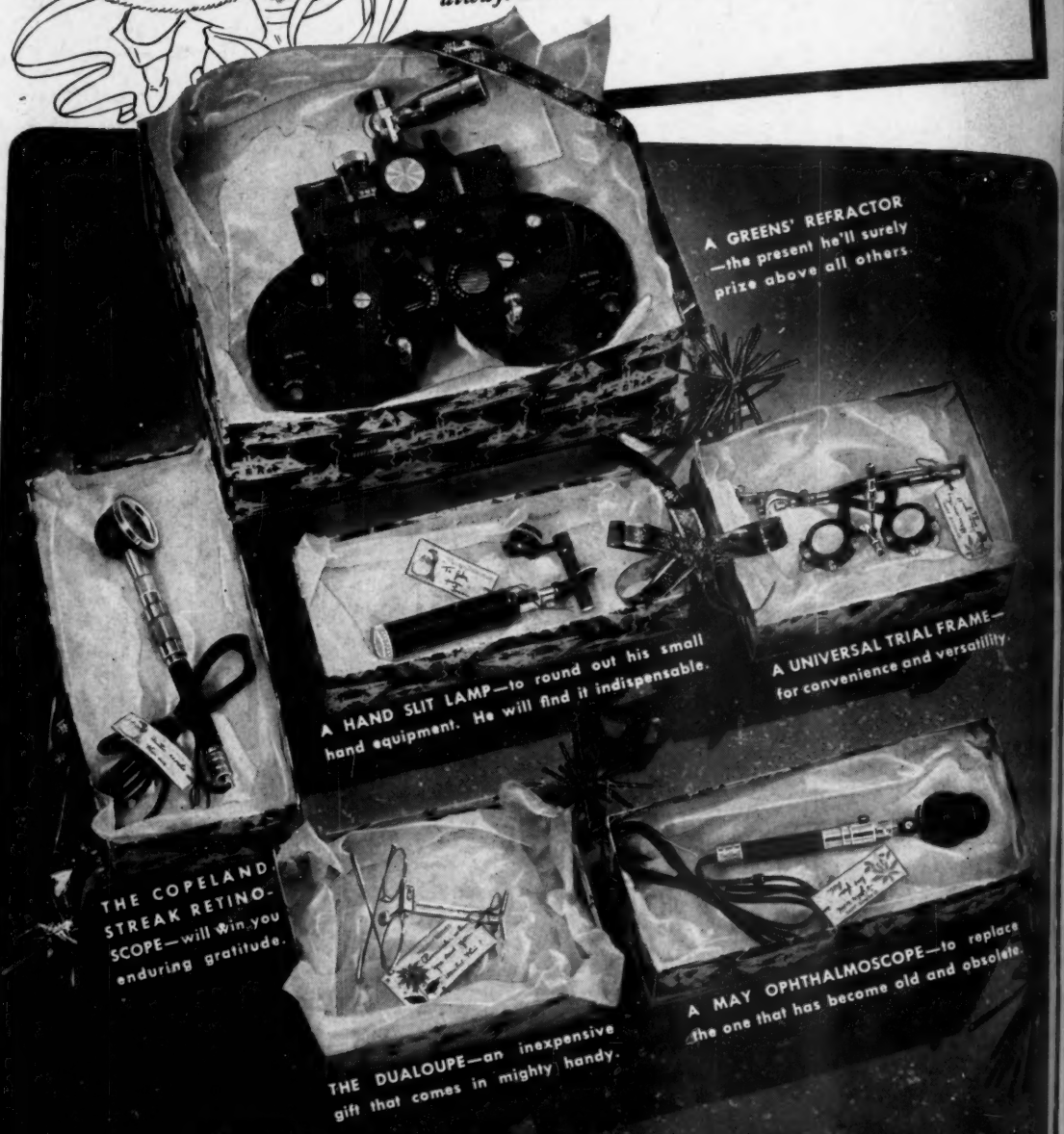
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